Investigating risk factors and prevalence for neurocysticercosis: a case study of Busia District, Kenya

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Declaration

I declare that the research described within this thesis is my own work and that this thesis is my own composition and certify that it has never been submitted for any other degree or professional qualification.

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Abstract

There has been a significant increase in pig production in the Eastern and Southern Africa region during the past decade (Githigia *et al.*, 2002), (Thuranira, 2005), especially in rural, resource-poor, small holder communities. Accompanying this has been the emergence of porcine cysticercosis as a problem in many of these areas including western Kenya (Mutua *et al.*, 2006).

Objectives: The objective of the study was to determine the prevalence of neurocysticercosis among epileptics in Busia District, Western Province, Kenya and investigate the risk factors associated with neurocysticercosis (NCC).

Methods: A group of 628 epileptics were identified using hospital and Special School records, key informant interviews and snowball survey techniques and a standard questionnaire to assess risk factors for neurocysticercosis or taeniasis, administered. Household information was also collected and an asset index formulated for each patient's household (n=471). Sera was taken from 630 subjects and tested for exposure to *T. solium* using an antigen enzyme-linked immunosorbent assay (Ag ELISA). The sera was also tested using an antibody (Ab) ELISA which tested for cysticercosis (metacestode exposure) and enzyme-linked immunotransfer blot assay (EITB, Western Blot) which tested for taeniasis and cysticercosis. Univariate and multivariate analysis was conducted to investigate the factors associated with seropositivity.

Results: There was one positive case of neurocysticercosis found by Ag ELISA and 209 subjects tested positive for exposure by Ab ELISA. There were 10 positive results using the EITB, 6 were positive using ES38 and 4 using Lentil Lectin purified glycoprotein (LLPG).

Conclusion: *T. solium* infections have multiple societal impacts including human health and productivity as well as livestock production and there needs to be further investigation into the burden of the disease.

List of Abbreviations

Ab	Antibody
Ag	Aantigen
ASF	African Swine Fever
BSE	Bovine Spongiform Encephalitis
CNS	Central Nervous System
EITB	Enzyme-Linked Immunotransfer Blot Assay
ELISA	Enzyme-Linked Immunosorbent Assay
GoK	Government of Kenya
ILRI	International Livestock Research Institute
LLPG	Lentil-Lectin Purified Glycoprotein
MoH	Ministry of Health

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Chapter 1: Introduction

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1.1 Introduction

Zoonotic cysticercosis is emerging as a serious problem in many developing countries in Latin America, Asia and Africa, causing economic livestock and human productivity losses, particularly amongst rural, poor, smallholder farmers (Phiri *et al.*, 2003). There are indications that with the increase in pig production in East Africa (Thuranira, 2005), porcine cysticercosis is also on the rise. In East Africa, porcine cysticercosis has been reported in Tanzania, (Nsengwa, 1995), (Ngowi *et al.*, 2004), Kenya (Githigia *et al.*, 2002), (Mutua, 2005) and Uganda (Anyanzo, 1999), (Mafojane *et al.*, 2003). Research in six districts in Western Kenya has shown evidence of porcine cysticercosis in all districts (Mutua *et al.*, 2006) with rates as high as 36% in Kimili Division using lingual examination – a method which is approximately 40% - 50% effective thus the true prevalence is probably much higher (Ngowi *et al.*, 2004). In Busia District in Western Kenya, prevalence rates for porcine cysticercosis were found ranging from 10-14% using the same diagnostic technique (Githigia, 2000). In Mbulu District, Tanzania, the prevalence rate was found to be 17.4% using lingual examination, so could in fact be as high as 35% (Ngowi *et al.*, 2004).

The disease is caused by the pork tapeworm *Taenia solium* which is a zoonotic parasite. The risk factors associated with the disease are common amongst poor, marginalised communities and include poor sanitation, free ranging pigs, lack of proper meat inspection or slaughtering facilities, and lack of health education concerning cysticercosis and other zoonotic parasite and helminth prevention strategies. In addition to cysticercosis, these communities are also subject to outbreaks of transboundary animal diseases such as African Swine Fever (ASF), a lethal, viral haemorrhagic disease of domestic pigs which has devastating economic consequences (Lubisi *et al.*, 2005). According to the International League Against Epilepsy (ILAE), human cysticercosis (neurocysticercosis) is probably the most common cause of acquired epilepsy in the developing world, where prevalence rates

of active epilepsy are twice those of developed countries (ILAE, 2006). Cysticercosis is important in smallholder farming communities because it exacts a price by:

- Causing infections in humans affecting their physical and psychological health, social life and productivity;
- Seriously reducing farmers' potential to participate in commercial markets for pig trading due to the barring of infected pigs by traders
- Compromising a valuable, relatively inexpensive source of protein in rendering pork unsafe to eat.

Pig keeping is a valuable tool for poverty alleviation in developing countries. In smallholder communities pigs are considered low-input livestock which do not require the same level of care or space as cattle, for example. In addition, pigs are omnivorous; and accomplished scavengers who can grow to market size on minimal feed inputs from the farmer. Thus cysticercosis is a serious constraint for improving the livelihoods for smallholder farmers in developing countries. There has been a significant increase in pig production in the Eastern and Southern Africa region (Githigia *et al.*, 2002) (Thuranira, 2005) during the past decade, especially in rural, resource-poor, smallholder communities. In Uganda and Kenya, the establishment of piggeries and increased pig production by rural farmers is encouraged by both respective governments forms part of the plan for the modernization of agriculture under the Ministry of Agriculture in Uganda and is part of the Busia District Development Plan, (Government of Kenya, 2002). In Uganda, the local governments are supplying piglets to the poor rural communities to rear in order to promote an alternative source of income.

Although cysticercosis in theory is relatively easy to prevent and control, many developing countries lack critical information concerning prevalence data, public health awareness of the problem, suitable diagnostic tools, surveillance systems and impact assessment data concerning the true burden of the disease.

This study aims to investigate and assess the burden of neurocysticercosis by establishing the prevalence rate of the disease in Busia District, Kenya and assessing the impact various risk factors have on acquisition of the disease.

The following section of the introduction provides background information on zoonoses - in general and with respect to poverty, porcine cysticercosis and neurocysticercosis and the various diagnostic and treatment options that are available for the disease. The next section examines neurocysticercosis-acquired epilepsy, focussing on its economic and social impact on people in the rural areas of developing countries. The subsequent section of the introduction looks at the importance of livestock keeping in general for smallholder farmers, disease control priorities in the developing world and an overview of helminth control strategies and *Taenia solium* control strategies specifically.

1.2 Zoonoses - general

In a comprehensive literature review of 1415 species of infectious organisms known to be pathogenic to humans, 868 or 61% are zoonotic. In the same study, of the 175 pathogenic species associated with diseases considered to be "emerging", 132 or 75% are zoonotic. Overall, zoonotic pathogens are twice as likely to be associated with emerging diseases than are non-zoonotic pathogens. Five major taxonomic divisions of pathogens were used within the study: viruses (including prions), bacteria (including rickettsia), fungi, protozoa and helminths (cestodes, nematodes, trematodes and acanthocephalans) (Taylor *et al.*, 2001). Three categories of transmission routes to humans were distinguished, these being: direct contact (including via wounds, sexual contact, vertical transmission or inhalation), indirect contact (via food or an environmental reservoir, and vector borne (biting or mechanical transfer by arthropods).

As the world's population continues to grow in an unprecedented fashion, emerging zoonotic diseases are amongst the most important public health problems facing humanity. One of the more prominent zoonoses to come to public attention in recent

years has been BSE or bovine spongiform encephalopathy and its human manifestation, Creutzfeld-Jakob disease. This particular outbreak disease occurred in a developed country and media attention focused largely on economic losses to farmers. There are many zoonoses, however, that occur in developing countries and go largely unnoticed as systems are not as highly developed for the monitoring of such diseases. While zoonoses themselves are difficult to diagnose (Pal *et al.*, 2000), in that they sometimes present few symptoms in both the animal and the human, even when symptoms are obvious, diagnostics are limited or prohibitively expensive (Dorny *et al.*, 2003). According to a report by the Secretariat of the World Health Organization which discussed policies and methods for the control of cysticercosis, "diagnostic criteria based on objective clinical, imaging, immunological and epidemiological data have been proposed for different levels of the health care system, but are not generally used in areas endemic for the disease" (WHO, 2002) In addition, there is often a lack of collaboration between the veterinary and medical organs of government resulting in information not being shared (Domingo, 2000). One of the greatest threats that zoonoses pose is in their ability to cross species barriers. Some of these diseases include influenza, human immunodeficiency viruses, filoviruses, Nipah virus and leptospirosis and more recently H1N5 avian flu and H1N1 swine flu (Pastoret *et al.*, 2000).

While traditionally it has been thought that rural livestock keepers were at most risk from zoonotic infections, with the increase in urban populations in developing countries expected to rise by 2030 to more than 50% of the population in Africa and Asia living in urban areas (Population Reference Bureau, 1999), diseases such as rabies (Cleaveland, 1998), water-borne zoonoses, and *T. solium* cysticercosis will be of particular importance. It is estimated that the number of people obtaining part of their food from Urban Agriculture in six East and Southern African countries will rise from about 25 million to 40 million by 2020. Urban Agriculture includes market garden vegetables but also livestock keeping such as cattle and poultry. The relationship between Urban Agriculture and the rural-urban transmission of zoonotic diseases remains under-researched, but as in many countries livestock keeping is not even officially allowed in urban areas, there is a clear need for urban policy for

delivery of human and animal health services to address this situation (UNHCS, 2001).

1.2.1 Zoonoses and Poverty

Zoonoses make up 61% of all species of infectious organisms known to be pathogenic to humans and are defined as those that are capable of "natural transmission between animals and humans" (WHO, 1959), thereby rendering their classification as zoonotic (Taylor *et al.*, 2001). In tropical regions, zoonotic diseases impose a considerable burden on human health and include, among others, sleeping sickness (Chambers & Conway), brucellosis, tuberculosis, hydatid disease, *Taenia solium* cysticercosis/taeniosis and rabies. In addition to their effect on human health, the impact of zoonoses extends to huge economic losses associated with the infections in the animal host (Coleman, 2002). As livestock and traditional production systems involving livestock are central to the livelihoods of the poor in developing countries (Delgado *et al.*, 1999); (LID, 1999), the poor are at risk both from the economic loss associated with being infected with the disease and the effects of the disease on their livestock (e.g. pastoralists and *M. bovis* exposure, (Moda *et al.*, 1996) and sleeping sickness and trypanosomiasis in Eastern Uganda (Fevre *et al.*, 2001).

The poor in every country bear a disproportionately high burden of disease (Gwatkin & Guillot, 2000; Sachs & Steele, 2001). According to the Food and Agriculture Organization of the United Nations (FAO),"although in the future there will be a dichotomy of issues and approaches in intensive versus traditional livestock systems, the impact of zoonotic diseases and food-borne infections and intoxications on health and wellbeing will be greatest among the 800 million food-insecure livestock keepers, consumers, traders and labourers" (FAO, 2002a). In the case of zoonotic disease, there are a number of reasons why their burden falls particularly heavily on poor people which go beyond the usual reasons of access, affordability and vulnerability.

Poor people are at more risk of contracting many zoonoses as they live in close proximity with their animals – the reservoirs of disease. Crowded living conditions, poor hygiene and sanitation services and practices characteristic of slums or shanties can increase the transmission of disease between humans, but also between animals and humans. In the case of zoonotic tuberculosis or *mycobacterium bovis*, human beings are infected from livestock kept for meat and milk production, especially bovines, but also including goats, sheep, buffalo and camels. The crowded slum areas around the larger cities in Asia, Africa and South America, where animals are kept in close proximity to humans, the transmission of this disease has the potential to increase drastically, should public health regulations not be enforced (Moda *et al.*, 1996). In addition, infected animal products are likely to be more appealing to the poor as they are sold more cheaply than uninfected, thus perpetuating the poverty and disease cycle. Examples of this include pork infected with cysts which cannot be sold legitimately at market, unpasteurised milk, meat from dying or infected animals sold off at slaughterhouses or the slaughter of animals that occurs in households without the presence of a meat inspector in the developing world (Mutua, 2005).

Evidence also indicates that poverty may be a significant risk factor in the seeking of proper treatment once infection is acquired. Zoonoses are often contracted by remote, marginalised populations making diagnosis a problem – lack of tools and facilities – and cost of transport to treatment facilities and the treatment regime itself becoming prohibitive. In an analysis of the determinants of vulnerability to malaria, tuberculosis and HIV, it was found that at the community and household level, malaria disproportionately affects lower socio-economic groups and that they are less likely to seek treatment or take preventive measures (Bates *et al.*, 2004). In sub-Saharan Africa, a diagnostic test for *Taenia solium* cysticercosis is available only in Zambia, making it virtually impossible for a smallholder farmer to have access to it, notwithstanding the procurement of the sample and the cost of transportation.

Poverty also contributes to the livestock themselves being more vulnerable to disease. According to a study which looked at the delivery of veterinary services to

the poor in Kenya, "few herders and farmers were spending close to the estimated 'ideal' on livestock drugs and the majority of expenditure was on curative rather than preventative treatments. Although apparently willing, the ability of the poor to pay for treatments appears to be a limiting factor. Knowledge regarding livestock health was poor, further contributing to the overall low uptake of veterinary goods and services. Both access and the quality of advice regarding the use of livestock drugs were considered problematic" (Heffernan & Misturelli, 2003). Many resource-poor farmers cannot afford or do not have access to public and private veterinary interventions or the necessary feed inputs to keep their animals in sanitary, disease-free conditions.

In a study carried out by the International Livestock Research Institute (ILRI) in 2002 (Perry *et al.*, 2002), diseases were ranked in order of those with the greatest importance to the livelihoods of the poor in each livestock productions system in their region. The diseases were then ranked by the identification and quantification of their impacts on the poor. Then, three major impacts of each disease or syndrome were identified and scored. These were socio-economic impacts (primarily production losses and control costs incurred by the poor), zoonotic impacts and national impacts (a combination of marketing impacts on the poor with public sector expenditure on disease control). A weighting was applied to the scores for each disease relating to the importance of different impacts on the poor (for example, socio-economic impact was given a weighting of 85% and national impact, 15%). Zoonotic diseases were ranked separately due to the difficulty of measuring the monetary value of human health impacts. The table below illustrates the top 20 diseases or pathogens ranked according to their impact on the poor, by species:

Table 1: Top 20 diseases ranked according to their impact on the poor, by species

	Buffalo	Cattle	Sheep/Goats	Poultry
TOP 10	Anthrax	Anthrax	Anthrax	Coccidiosis
	Brucella abortus	Brucella abortus.	Ecto-parasites	DVE
	Foot and Mouth Disease	CBPP	Haemonchosis	Ecto-parasites
	Haemorrhagic septicemia	Foot and Mouth Disease	Heartwater	Fowl cholera
	Liver fluke	Haemorrhagic septicemia	Helminthosis	Fowl pox
	Reproductive disorders	Liver fluke	Liver fluke	Helminthosis
	Respiratory complexes	Nutritional/micronutr def	Neonatal mortality	Infectious Coryza
	Rinderpest	Reproductive disorders	PPR	Neonatal mortality
	T. evansi	Toxocara vitulorum	Respiratory complexes	Newcastle Disease Virus
	Toxocara vitulorum	Trypanosomosis	Sheep & goat pox	Nutritional/micronutr def
NEXT 10	Blackleg	Babesiosis	Blue tongue	DVH
	Bovine tuberculosis	Blackleg	Brucella melitensis	Gumboro
	Buffalo Pox	Dermatophilosis	CCPP	Mycoplasmosis
	Diarrhoeal diseases	Diarrhoeal diseases	Clostridial diseases	Salmonella
	Mastitis	Helminthosis	Foot and Mouth Disease	
	Nutritional/micronutr def	IBR	Foot problems	
		Mastitis	Orf	
		Neonatal mortality	Para-tb	
		Rinderpest	Rift Valley fever	
		Theileria annulata	Trypanosomosis	

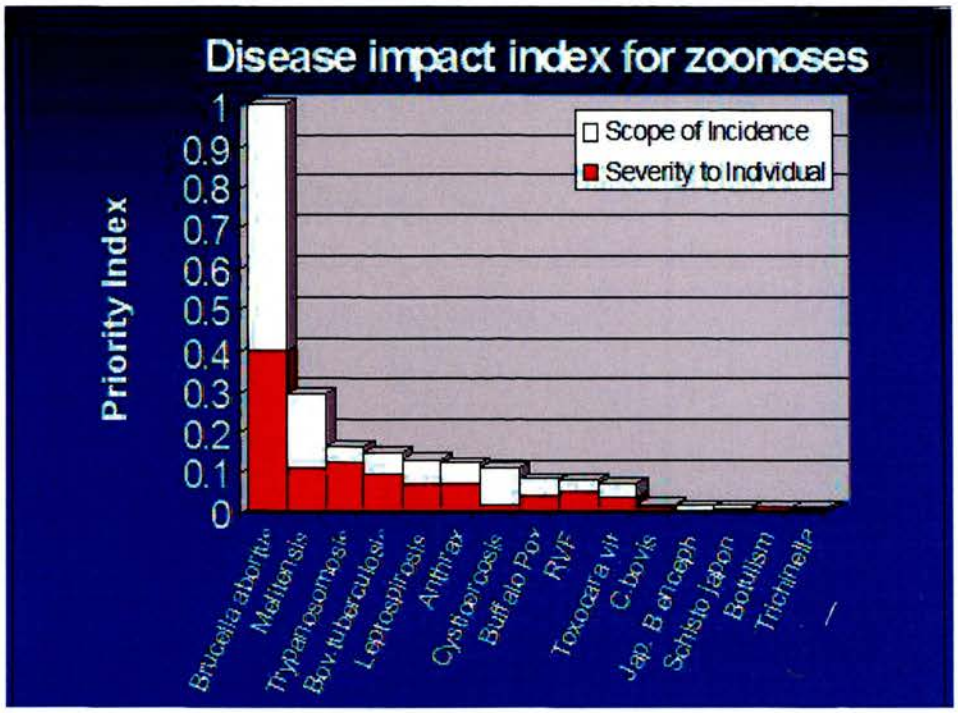
	Pigs	Camels	Donkeys	Yaks
TOP 10	ASF	Acute Resp Syndrome	Helminthosis	Foot and Mouth Disease
	Brucella suis	Camel Pox	Trypanosomosis	Haemorrhagic septicemia
	Cysticercosis	Haemonchosis	Wounds/Injuries	Liver fluke
	Ecto-parasites	Helminthosis		Neonatal mortality
	Foot and Mouth Disease	Mange		
	Helminthosis	Neonatal mortality		
	Hog cholera	Respiratory complexes	**Horses**	
	Neonatal mortality	Rift Valley fever		
	Japanese B encephalitis	T. evansi	AHS	
	Trypanosomosis	Tick Infestation	Lymphangitis	
		Anthrax		
		Rabies		

Source: (Perry *et al.*, 2002)

The two graphs that appear below illustrate the disease impact that zoonoses have both in terms of scope of incidence and severity of impact to the individual. The first graph represents an overall world-wide perspective, while the second is divided by region. It is acknowledged that diseases that impact livestock productivity, the

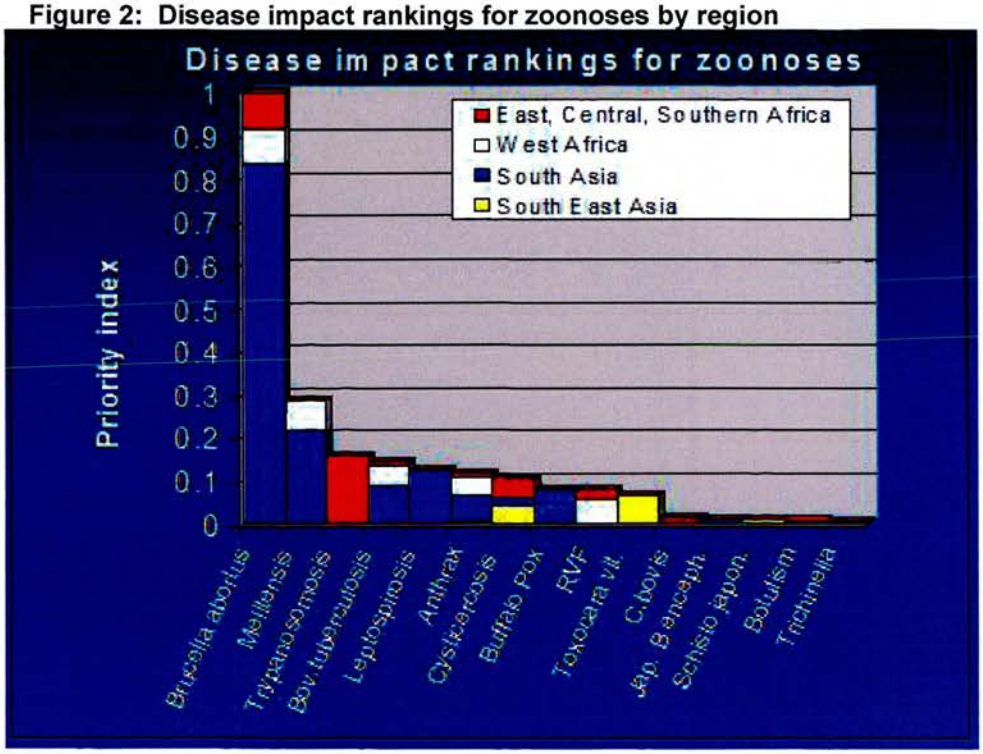
marketing of livestock and their products, and human health, have greater impacts on poverty than those having an impact on only one of these areas (Perry *et al.*, 2002).

Figure 1: Disease impact index for zoonoses



Source: (Perry *et al.*, 2002)

Figure 2: Disease impact rankings for zoonoses by region



Source:(Perry *et al.*, 2002)

1.2.2 Zoonotic Disease Control: A question of "Divided Constituencies"

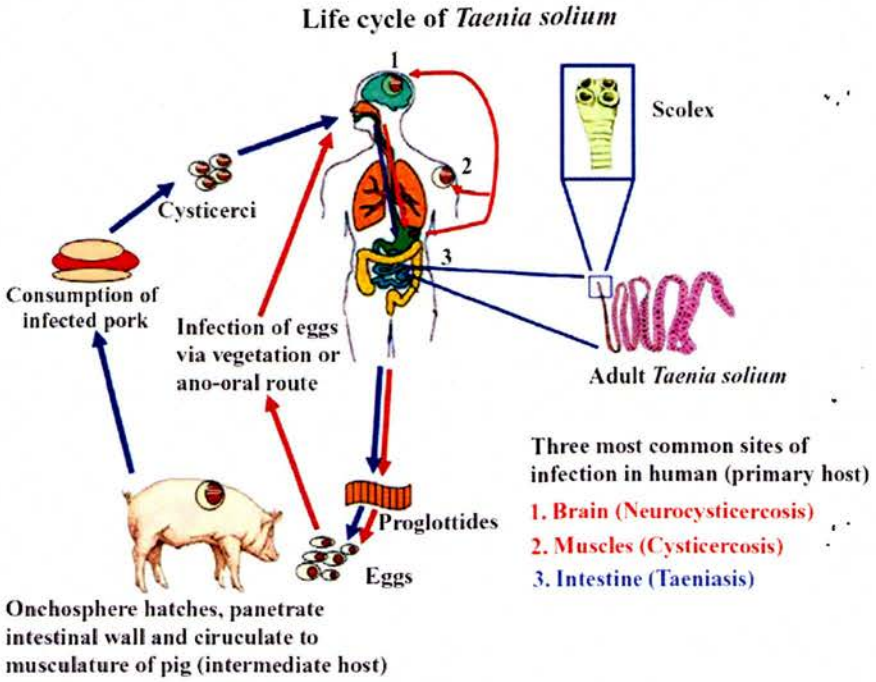
One of the problems facing a unified strategy for zoonotic disease control has been that medical and veterinary professionals and sectors have traditionally focussed on the improvement of human health and on livestock production as their primary but separate objectives (WHO, 2006). Zoonoses often fall, overlooked, into the gap between veterinary responsibities and medical needs. For many zoonoses, it is the risk to human health that is considered of greatest importance, while the most effective control route is through the animal. It is clear that these two sectors need to work in tandem in order to operate most effectively, however, there needs also to be the appropriate mechanisms in place to support this effort. These include strong legal and regulatory frameworks to help enforce disease prevention and control laws and regulations such as meat inspection and vaccinations. Policy frameworks for both sectors must also adopt a unified approach to human and animal disease control.

1.3 Cysticercosis

1.3.1 History

Cysticercosis was originally a ubiquitous disease occurring wherever pigs and humans existed in association, and is probably a disease of great antiquity – Aristotle gives a clear description of the condition as it occurs in pigs (Cook & Zumia, 2003). Although cysticercosis has been known for many years, its relationship with the adult tapeworm appears not to have been clear until it was demonstrated by Kuchenmaister in 1855 when he fed condemned prisoners with cysticercosis-infected pork and recovered young tapeworms at the autopsies (Henneberg, 1912). There is, however, some evidence to suggest that as early as the 6th century there may have been some knowledge about it as illustrated by the example of Anthinus who reported to Theodoric, King of the Franks (511-534), that "tapeworms were excreted after the consumption of raw fatty pork" (Gach, 1926). Even if this connection had been established, there is no evidence that a link was made between the tapeworm transmitted and its effects on humans.

Figure 3: Life Cycle of *Taenia solium*



Source: (Prasad *et al.*, 2008)

1.3.2 Life Cycle of *Taenia solium*

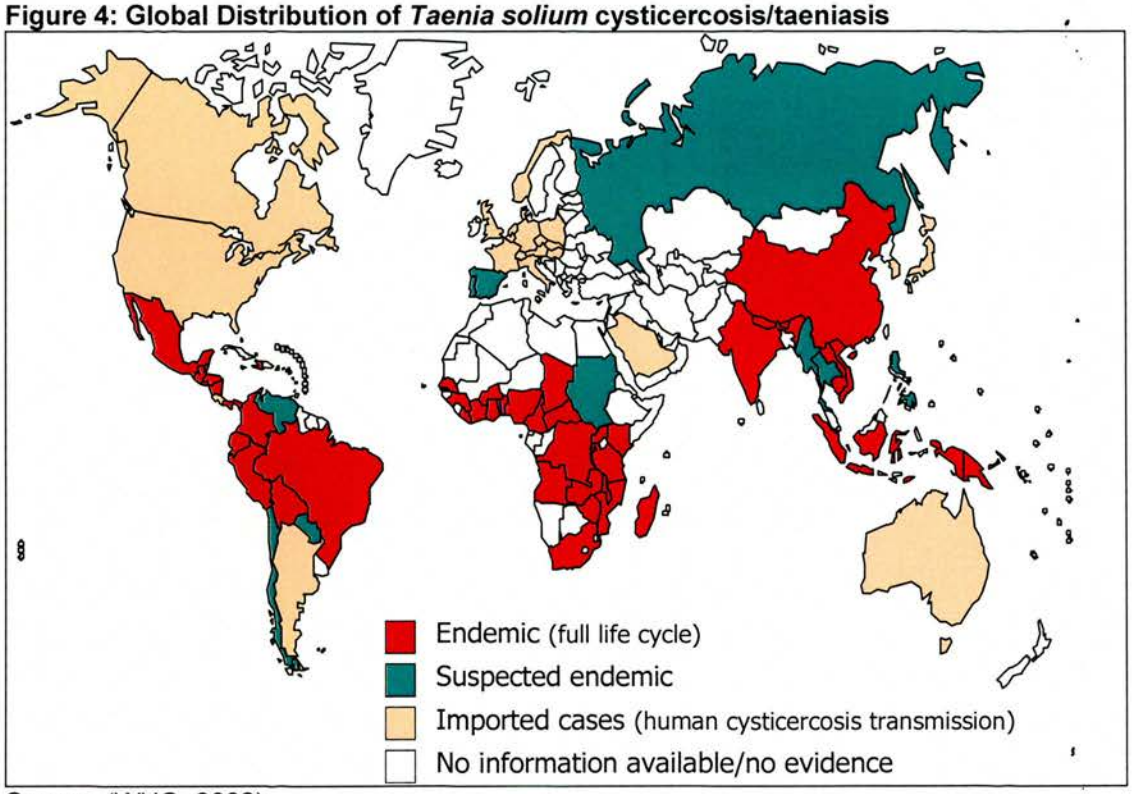
T. solium has a complex two-host life cycle. Human beings are the only definitive host and harbour the adult tapeworm (taeniosis), whereas both people and pigs can act as intermediate hosts and harbour the larvae or cysticerci (Garcia et al., 2003a). When a human eats contaminated pork which contains cyst, they develop into adult worms in the small intestine. The adult worm attaches itself to the mucous membranes in the intestines by a scolex which is equipped with suckers and a rostellum which contains 25-50 hooklets. The proglottids which are full of eggs are discharged into the environment by excretion of faeces by the human. The eggs are spherical in shape and extremely small (30-40 μm in diameter). After they are free from the proglottids, the eggs can then be ingested by pigs, man or other animals. Once in the intestine, the protective coating around the eggs is broken down by gastric and pancreatic enzymes and embryos or oncospheres are released. Aided by their hooklets, the oncospheres cross the intestinal wall and local venules, enter systemic circulation and are carried to different organs of the host (skeletal muscles, central nervous system (CNS), subcutaneous tissue, eye, etc.). Here the oncospheres

lose their hooklets, acquire a vesicular shape and evolve into cysticerci by gradual evagination of the protoscolex (invaginated scolex) over a period of two months (Escobar and Neito 1972). The life cycle is completed when undercooked pork infested with cysticerci is eaten by human beings. It is possible, however, for someone who has never eaten pork to contract NCC. Man may also become an intermediate host and develop the larval stage of the disease in one of three ways: (i) by heteroinfection, the most common route, in which eggs present in food contaminated by the faeces of *Taenia* carriers are ingested; (ii) by exogenous autoinfection, due to ano-oral contamination in patients harbouring the adult worm; (iii) by endogenous autoinfection, in which the eggs of the adult tapeworm living in the small intestine return to the stomach due to reverse peristalsis. The last two modes of autoinfection are far less frequent as it is quite uncommon to find patients having simultaneous infestation with cysticercosis and taeniasis in more than 10-15% cases (Rabiela-Cervantes *et al* 1982). In humans, the parasite can become lodged in the Central Nervous System, resulting in NCC (Del Brutto and Satelo 1988). The larval stage also infests other tissues like skeletal muscle, diaphragm, heart and peritoneum, pleura and subcutaneous tissue (Shankar *et al* 1994). Mammals other than swine have also been reported to harbour cysticerci of *T. solium*. In Indonesia, examination of two sero-positive dogs revealed cysticerci of *T. solium* in their brain and heart (Ito *et al* 2002). *C. cellulosae* have also been recovered from the brain of a cat (Schwan *et al* 2002).

1.3.3 Geographical Distribution

Taenia solium is found worldwide. Because pigs are intermediate hosts of the parasite, completion of the life cycle occurs in regions where humans live in close contact with pigs and eat undercooked pork. Taeniasis and cysticercosis are very rare in Muslim countries, but it is important to note that human cysticercosis is acquired by ingesting *T. solium* eggs shed in the faeces of a human *T. solium* tapeworm carrier, and thus can occur in populations that neither eat pork nor share environments with pigs (CDC, 2004).

Figure 4: Global Distribution of *Taenia solium* cysticercosis/taeniasis



Source: (WHO, 2002)

1.4 Porcine cysticercosis

In East Africa, the porcine cysticercosis has been reported in Tanzania, Kenya and Uganda. In Tanzania, a retrospective study of slaughter records in Mbulu district from 1985-1989 indicates a prevalence increasing from 0.4 to 4.9% during that time (Nsengwa, 1995). In a more recent study of the risk factors associated with cysticercosis in Mbulu District, pigs raised in a total of 436 households were examined in the 21 selected villages in the district. Out of the 770 pigs examined, 134 had visible *T. solium* larvae, giving an overall prevalence of 17.4% (95% CI = 12.5, 22.3). The prevalence in the 21 villages ranged from 3.2 – 46.7%, showing a large variation in villages (Ngowi et al., 2004).

In Kenya, the disease has not been considered endemic for several decades but recent surveys indicate that *T. solium* is emerging as a problem in small-holder pig keeping communities of south-western Kenya where ante-mortem lingual examinations in different locations in the area have indicated at least 10-14% of the pigs surveyed to be infected (Githigia et al., 2002). According to the Annual Reports of the Ministry

of Health (MOH), the prevalence of human taeniosis in the rural areas is estimated to be 2% (Mafojane, 2003) but could be as high as 4-10% in Busia District as hospital records suggest (Busia District Hospital, 2001).

In Uganda, there is not enough data to quantify the prevalence of cysticercosis, but according to a post-mortem survey conducted in 1999 in northern Uganda, 34-45% of the pigs slaughtered were infected. Given the increases in pig-keeping in Uganda over the past 20 years and the prevalence of porcine cysticercosis in neighbouring countries, it is likely that there will soon be a public health problem (Mafojane, 2003).

The disease has been reported in West African countries such as Benin, Burkina-Faso, Ghana, Ivory Coast, Senegal and Togo and is expected to be present in most pig-raising regions of other West African countries. In the Nsukka area of Enugu State in Nigeria, over 20% of 2358 trade pigs examined were found infected with porcine cysticercosis and the overall prevalence of taeniid ova in the 1525 human stool samples analysed was 8.6% with most of the cases (78.6%) occurring adults aged >30 years (Onah & Chiejina, 1995). Although economic losses are associated with the decreased value of a pig infected with cysticercosis (Zoli et al., 2003a), it has to be noted that in some villages in West Cameroon, pork infected with cysticerci is considered to have a better flavour than healthy meat. Therefore, pork harbouring cysticerci is sometimes sold at a higher price than uninfected meat (Zoli & Tchoumboue, 1992).

1.4.1 Diagnostic Tools for Porcine Cysticercosis

1.4.1.1 Lingual Palpation

For detection of pigs infected with cysticerci of *T. solium*, there is a well-known traditional method involving examination of the pig's tongue. It is based on the identification of putative parasitic lesions in tongue muscles of pigs. A pig is restrained in lateral recumbency and the head stabilized by the use of a pig snare. The mouth is opened by the aid of a wooden rod twisted across the upper and lower jaw and the tongue gently pulled out using a piece of a cotton cloth. The under-

surface of the tongue is examined for the presence of *T. solium* larvae. Mature viable *T. solium* larvae are oval, fluid-filled, and with a central whitish spot, which is the protoscolex. Immature larvae are smaller, fluid-filled and no protoscolex can be distinguished. The fluid content is absent in degenerated cysts and such cysts can easily be confused with other conditions such as abscesses (Ngowi et al., 2004). This is the most common way for ante-mortem detection of infected pigs in the majority of developing countries where NCC is endemic (Gonzalez et al., 2001; Ito et al., 2003a), but as previously described, does require a certain level of technical skill. Although tongue palpation is reported to be highly specific, it is only moderately sensitive, especially in light infections (Gonzalez et al., 1990). In a more recent study in Zambia comparing the specificity and sensitivity of tongue inspection and carcass inspection, the specificity of tongue inspection was 100% but both methods failed to detect the infection in 83.9% and 61.3% of infected pigs, respectively, thus rendering the sensitivity very low (Phiri, 2006). According to a study in which a Bayesian approach was used to estimate the sensitivity and specificity of various diagnostic tests, the sensitivity of tongue inspection was 21% and the specificity 100% (Dorny *et al.*, 2004). In a study conducted in Brazil, none of the experimentally infected animals slaughtered after 140 days of the infection showed clinical aspects of cysticercosis or cysts by tongue palpation (Gomes, 2007). These studies further illustrate the degree to which swine with a low cyst burden can be missed by palpation and the extent to which the prevalence of cysticercosis in pigs in endemic regions may be underestimated.

1.4.1.2 Post-mortem carcass inspection

Post-mortem meat inspection for slaughter pigs with respect to cysticerci of *T. solium* generally relies on visual examination of different sites in the carcass considered to be predilection sites for the parasite including the heart, diaphragm, masseters, tongue, neck, shoulder, intercostal and abdominal muscles (Gracey, 1986). The masseters, followed by the pterygoid and the triceps muscles, have been found to have the highest number of cysticerci per 100g and are thus determined to be predilection sites (Mendez, 1986). One of the factors affecting detection of cysts is

the density of cysts which can determine whether the cysts are visible to the naked eye during inspection.

While this method is considered effective in determining infected pigs, it relies heavily on farmers bringing their animals to be inspected at government inspection facilities. In the rural areas of many African countries it is very difficult to enforce inspection as slaughter inspection points are not easily accessible (personal observation) leading many farmers slaughter to within their homesteads and consume the meat in situ. In addition, in some countries such as Tanzania, pigs are generally slaughtered in the early hours of the morning when light for inspection of dressed carcasses is often inadequate (Boa *et al.*, 2002). It has also been noted that when farmers suspect their pigs to be positive for cysts, they tend to slaughter their pigs at home and eat the pork at home or share with neighbours or later sell the meat through informal channels rather than risk their condemnation at the slaughter facility (The Cysticercosis Working Group in Peru, 1993). Although meat inspection is a viable means of cyst detection, the fact that many carcasses escape inspection due to a variety of reasons renders this method less effective as a means of detecting porcine cysticercosis within a population. One of the reasons that prevents necroscopy from being frequently conducted, especially in regions where poverty is endemic, is that the best routinely used exam requires several meat-screening cuts, damaging the product for commercial purposes and thereby decimating the income of the prospective farmer or trader (Gomes, 2007). The performance of this test in Zambian local pigs was 22% sensitive and 100% specific, a statistic similar to that of tongue inspection (Dorny *et al.*, 2004).

1.4.1.3 Enzyme-linked Immunoelectrotransfer Blot (EITB)

This test is derived from the EITB developed for the immunodiagnosis of human cysticercosis (Tsang *et al.*, 1989). It is based on the identification of specific glycoproteins and uses either the *Taenia solium* cysticercus antigen prepared by purification on lentil-lectin sepharose and ultracentrifugation which is difficult to obtain in many developing countries (Pathak *et al.*, 1994) or a crude *Taenia solium* extract which is easier to prepare (Gottstein *et al.*, 1986). In developing countries,

ELISA is preferred because of its better availability, simplicity and lower cost compared with immunoblot (Rosas *et al.*, 1986).

The EITB with crude *Taenia solium* metacestode extract has been shown to have a sensitivity of 90% and a specificity of 100% (Pathak *et al.*, 1994), however, in that particular study, the number of pig sera tested was low (20 pigs). The EITB glycoprotein technique employed by Tsang et al. (1989) was evaluated and produced a sensitivity and specificity of 100% (Gonzalez *et al.*, 1990). The laborious and expensive purification of glycoprotein antigen from the cysticerci, however, makes the former method using crude cysticercus antigen more feasible and practical for laboratories in developing countries. The lentil-lectin-purified glycoproteins from *T. solium* Ag (LLGP) EITB has proved more useful in determining infected pigs with a very light cyst burden (86% positive) than has the Ag ELISA (57% positive) and the Ab ELISA, while the EITB using fluid from *T. crassiceps* Ag proved as effective diagnostically as the Ab ELISA (Basanez; Basanez *et al.*, 2004) (Gomes, 2007).

1.4.1.4 Enzyme-linked immunosorbent assay for the detection of specific antibodies (Ab ELISA)

Immunoassays for antibody (Ab) detection can be used prior to slaughter, primarily to isolate the non-infected animals from those more likely to be infected (Gomes, 2007). In pigs, the benefits of immunodiagnosis are:

i) tests offer diagnosis on live animals;

ii) blood sampling followed by serological testing is more sensitive than the classical tongue examination; and

iii) the tests are relatively inexpensive and easy to perform on large numbers of serum samples (Dorny *et al.*, 2003).

In a study carried out in India using the infected carcasses of pigs and cysts transported from the UK, sera from 20 pigs with parasitologically confirmed cysticercosis were tested by both EITB and Ab ELISA assays. Specificity of the tests was examined by testing 25 serum samples from the UK, where cysticercosis is not endemic and sera from pigs with *Echinococcus granulosus (15), Fasciolopsis buski*

(six), or *Trichinella spiralis (five)* infections. All but two of the samples from cases of confirmed cysticercosis were positive by EITB and none of the samples from healthy controls or heterologous infections reacted to any of the diagnostic bands. Thus, the test was 90% sensitive and 100% specific. The sensitivity of the ELISA was 70% with 73% specificity, cross-reactions occurring with sera from *E. granulosus* infected pigs. Four polypeptides (8, 11, 16 and 23 kDa) were identified by SDS-PAGE and EITB that were specifically recognized by pigs with confirmed cysticercosis (Pathak *et al.*, 1994).

1.4.1.5 Enzyme-linked immunosorbent assay for the detection of specific antibodies (Ag ELISA)

The Ag ELISA used to determine porcine cysticercosis was initially developed for *T. saginata* cysticercosis (Brandt *et al.*, 1992) and is also used to determine human cysticercosis

1.4. 2 Treatment

The treatment of porcine cysticercosis is potentially beneficial to farmers, but in many communities, farmers regard their pigs as zero-input animals and are reluctant to spend money on curing the pig. Many pig owners are not interested in knowing the parasitic burden the animal carries and will slaughter and eat the pig, regardless of whether it is sick or not (Rodriguez-Canul *et al.*, 1998).

Human anti-helminthics have been used in treatment of porcine cysticercosis with some success. These include albendazole, praziquantel, flubendazole and oxfendazole (Flisser *et al.*, 1990), (Gonzalez *et al.*, 1995), (Gonzales *et al.*, 1996). Albendazole and praziquantel drugs, however, have produced some side effects in pigs, including anorexia, fatigue and lethargy (Gonzalez *et al.*, 1995) and are prohibitively expensive to a smallholder farmer. In 2001, the price of albendazole, flubendazole or oxfendazole ranged from US$ 1.20 to 1.60 per dose (Gonzales, 1998), (Gonzalez *et al.*, 2001).

An alternative to these drugs could be the use of an injectable, specifically veterinary drug such as albendazole sulphoxide (ABZSO). The use of this drug has been evaluated in a chemotherapy study carried out in Mexico (Peniche-Cardena *et al.*, 2002). In this study, seven naturally infected pigs from 6 to 12 months of age were divided into 2 groups – treated (n=4) and control (n=3). The treated animals received a subcutaneous injection in their forelegs and thighs of 15 mg/kg per body weight of albendazole sulphoxide (ABZSO; Pisa, Mexico) once per day for 8 days. At the same time, the control group received a subcutaneous injection of saline solution (9% NaCl). After 12 weeks, all the animals were slaughtered and at least 200 metacestodes were isolated from the muscles and brain of each animal. It was found that treatment of pigs with a 15 mg/kg dosage of subcutaneous ABZSO (Pisa, Mexico), applied daily for 8 days was 100% effective in the control of muscle cysticercosis. This dosage was insufficient, however, to affect the morphology and viability of cerebral cysticercosis. There was evidence that the parasites were killed, however, more time is needed for complete disappearance of the degenerated cysts from the animal. The study concluded that the potential for use of ABZSO is encouraging. The price of the drug (US$ 0.20 per dose) should not be a hindrance as it is 100% effective in treating muscle cysticercosis and would allow farmers to limit their economic losses.

1.5 Taeniasis

Taeniasis is the stage of human infection which occurs after ingestion of raw or poorly cooked meat which is either infected with *Taenia solium* cysticerci or *Taenia saginata* cysticerci. In the life cycle, only humans are capable of harbouring the adult tapeworm, whereas both humans and pigs may act as intermediate hosts and harbour the larvae or cysticerci (CDC, 2004). This stage involves the larvae evaginating in the small intestine and the head or scolex attaching to the mucous in the intestine. Adult tapeworms then begin to develop over a two month period and can be from 2 to 7 m in length. These tapeworms form approximately 1000 proglottids (each carrying around 50,000 to 60,000 eggs) which can detach from the distal end of the worm and be excreted in the faeces of the carrier (at a rate of approximately 6 per

day) leaving the eggs to be eaten by pigs and other animals thus ensuring the cycle continues (CDC, 2004).

1.5.1 Clinical Presentation

The adult tapeworm can live in the small intestine for many years with few or mild symptoms (Schantz *et al.*, 1998).

1.5.2 Diagnostic Tools for Detection of Taeniasis

1.5.2.1 Coproantigen

Classically, taeniasis has been detected by direct parasitologic examination of stool samples. Detection methods, based on microscopic observation of eggs or proglottids in feces, are neither sensitive nor specific (Diaz *et al.*, 1992a),(Schantz, 1994). Direct examination and detection of *Taenia* eggs is dependent on the skill of the technician and requires examination of expelled proglottids for this distinction (Eldson-Dew & Proctor, 1965). A coproantigen detection assay has been developed and has been found to greatly increase the sensitivity of detecting taeniasis cases (Allan *et al.*, 1990; Allan *et al.*, 1992). This assay, which can detect parasite antigens expelled in stool, has been used successfully in field situations (Allan *et al.*, 1993) and was shown to be 99% sensitive and greater than 99% specific for *Taenia sp.* One of the few disadvantages of this technique is that it is unable to distinguish *T. solium* and *T. saginata* infections (Allan *et al.*, 1990).

1.5.2.2 DNA Probes

A method using DNA probes specific *for T. solium* or *T. saginata* was developed that uses species-specific primers to differentiate these two tapeworm infections (Harrison *et al.*, 1990; Chapman *et al.*, 1995). This technique relies on the amplification of parasite DNA obtained from parasite eggs or proglottids present in the stool sample.

Although the polymerase chain reaction can detect the presence of a single egg (Chapman *et al.*, 1995), the fact that passage of the eggs in the stool is erratic limits the usefulness of this assay.

Regardless of the detection method used, the collection and examination of stool samples for identification of taeniasis cases is associated with several problems. Primarily, collection of fecal samples carries with it the potential for exposure to and infection with *Taenia* eggs present in the sample and the sample must be tested within a specific time period for it to remain viable. In addition, collection of stool samples is often difficult and culturally unacceptable in many places where epidemiologic studies on cysticercosis and taeniasis are conducted (Wilkins *et al.*, 1999).

1.5.2.3 EITB for Taeniasis

Humans can be infected with both the adult worm and larval forms of the cestode *Taenia solium,* causing taeniasis and cysticercosis, respectively. Identification and treatment of taeniasis infections can be difficult to detect since they are usually asymptomatic. Therefore, accurate and sensitive detection of taeniasis cases becomes a critical element for developing successful control strategies for cysticercosis (Schantz *et al.*, 1993; Gilman *et al.*, 1996).

A serologic EITB assay for detection of taeniasis in humans has been developed by (Wilkins *et al.*, 1999) which uses excretory/secretory (ES) products from *T. solium* tapeworms (TSES) that detect tapeworm carriers in a sensitive and specific manner. The specificity of the TSES immunoblot assay has also been examined with respect to differentiation of *T. solium* and *T. saginata* infections. There were no cross-reacting antibodies present in any *T. saginata* samples that recognized any TSES antigens, including the TSES diagnostic antigens. In order to examine the performance of the assay with respect to cross reactions with other parasite diseases, serum samples from patients with other parasitic diseases were also examined for antibodies to the TSES antigens (Table 2). None of the 193 samples examined

contained antibodies that reacted with the target ES antigens. The serum battery included 69 serum samples from patients with echinococcosis, and seven serum samples from patients infected with *Hymenolepis nana.* Some serum samples from echinococcosis patients contained antibodies that reacted with other higher molecular weight antigens in the ES mixture, but not with the diagnostic antigens, indicating the assay is 100% specific. Using serum samples collected from persons with confirmed *T. solium* tapeworm infections, the test was determined to be 95% (69 of 73) sensitive (Wilkins *et al.*, 1999).

Table 2: ES diagnostic proteins for effectively detecting only *T solium* taeniasis

Specificity of the *Taenia solium* ES diagnostic proteins for detecting only *T. solium* taeniasis

Infection*	No. of samples tested	No. of samples positive
T. solium infections		
Taeniasis	73	69
Cysticercosis	23	1
Other cestode infections		
T. saginata taeniasis	8	0
Echinococcosis	69	0
Hymenolepis nana	7	0
Non-cestode infections		
Schistosomiasis	37	0
Filariasis	30	0
Ascariasis	30	0
Trichinellosis	4	0
Drancunculiasis	4	0
Protozoal infections		
Amebiasis	4	0

* Filariasis sera were collected from individuals infected with onchocerciasis (n = 26) and lymphatic filariasis (n = 4, caused by *Wuchereria bancrofti*). Schistosomiasis infection sera were collected from persons with *S. mansoni* (21), *S. haematobium* (8), and *S. japonicum* (8) infections.

Source: (Wilkins *et al.*, 1999)

1.6 Neurocysticercosis

Neurocysticercosis (NCC) is a parasitic infection that can affect the nervous system caused by *Taenia solium,* which is common, but preventable. In both humans and pigs, a therapeutic does of drugs is the most common form of treatment but often detection is under-reported (Tsang & Wilson, 1995) and better surveillance and

monitoring is necessary to control the infection. Infection with *T. solium* is widely prevalent in human and pig hosts in many developing countries of Latin American, Africa, and Asia (Sarti & Rajshekhar, 2003).

Humans are the only natural definitive host while pigs are the intermediate host. Man may become an intermediate host from ingestion of the eggs of the adult tapeworm, resulting in a condition known as human cysticercosis. The cysticerci of *T. solium* may lodge in the brain causing cerebral cysticercosis (neurocysticercosis), resulting in headaches, epileptic seizures, blindness and mental disturbances (White, 2000). In addition, the cysticerci may cause other neurological afflictions such as meningitis, encephalitis, ventricular disease, spinal disease and ocular cysticercosis, although the most common manifestation still remains epilepsy (Cook & Zumia, 2003).

1.6.1 Diagnosis of Neurocysticercosis

According to the diagnostic criterion set out in "The Current Consensus Guidelines for the Treatment of Neurocysticercosis" (Garcia 2002), patients who exhibit cystic lesions showing the scolex of the parasite on a CT scan or an MRI fit the "absolute" criterion. The presence of cysts in the brain indicates a definitive diagnosis.

Definitive diagnosis – these are patients who have one absolute criterion or those who have two major plus one minor and one epidemiologic criterion, or
Probable diagnosis – those patients who have one major plus two minor, those who have one major plus one minor and one epidemiologic condition, and those patients who have three minor plus one epidemiologic condition (Del Brutto 2001).

1.6.2 Diagnostic Tools for Neurocysticercosis

According to (Dorny *et al.*, 2003), antibody detection tests (e.g. ELISA and EITB) are the most appropriate tools for measuring exposure to *T. solium* in sero-epidemiological surveys and for confirmation of *T. solium* as the etiological agent of epilepsy, but antigen detection tests are more useful for other purposes such as the detection of active cysticercosis or the follow-up of NCC patients after treatment. As

Ag ELISAs only detect cases of active cysticercosis (i.e. the presence of living cysticerci) (Erhart *et al.*, 2002), (Garcia *et al.*, 2000), (Nguekam *et al.*, 2003), which is important for deciding on the appropriate antiparasitic treatment (according to the guidelines proposed by (Garcia *et al.*, 2002)), when identifying *T. solium* as the etiological agent of epilepsy, antibody detection is more appropriate than Ag-ELISA because dead cysts are more-often responsible for epileptic seizures than those caused by living cysts (Zoli *et al.*, 2003b). However, in sero-epidemiological studies, antibody detection tends to overestimate the prevalence of cysticercosis because antibodies are no longer detected in serum within 1–3 years in 30–40% of patients sero-positive for *T. solium* in endemic countries (Garcia *et al.*, 2001). This reflects a transient antibody reaction in patients after exposure to *T. solium* eggs or is a result of self-cure by the patient.

A study was carried out by (Prado-Jean *et al.*, 2007) to evaluate the benefits of the detection of both circulating antibodies (Ab) and antigens (Ag) for the diagnosis of cysticercosis in people with epilepsy. Serum samples were collected from subjects in a matched case–control study for epilepsy in the Kiremba area, Burundi, between March and April 2001 (epileptic cases ¼ 303; controls without epilepsy ¼ 606). The enzyme-linked immunosorbent assay (ELISA) was used for the detection of antibodies (Ab ELISA) and circulating Ag (Ag ELISA). The Ab ELISA revealed 58.7% positivity in epilepsy cases and 31.4% in healthy controls; and Ag ELISA revealed 38.3% positivity in epilepsy cases and 20.0% in controls. The matched odds ratios were 3.6 (95%CI: 2.5–4.9) for Ab-ELISA, and 2.9 (95%CI: 2.1–4.3) for Ag ELISA. Both Ag and Ab ELISA detected a significantly higher number of seropositives among people with epilepsy than among controls. The risk of epilepsy was high in cases with a positive Ag ELISA, although less important than in cases with positivity for Ab ELISA. Dead or degenerating cysticerci appear to be more frequently associated with epilepsy than living cysts.

(Ito & Craig, 2004) continue the debate about the efficacy and purpose of antibody and antigen tests, as they claim that antibody assays are suitable for detecting cases

of active cysticercosis with or without a history of epilepsy. This is also supported by data obtained from tests involving blind controls in humans (Wandra *et al.*, 2003), pigs (Sato, 2003) and dogs (Ito *et al.*, 2002). More detailed molecular information on candidate T. solium antigens in order to develop a suitable Ag-ELISA and further research, in addition to phenotypic work on cysticercosis, should resolve this issue.

1.6.2.1 EITB

Currently, the most reliable immunological test for the diagnosis of NCC is an EITB assay with serum samples using Lentil Lectin Glycoprotein (LLGP) antigens extracted from the metacestode of *T. solium* and is reported to have a high specificity (100%) for the detection of antibodies in serum and CSF (Tsang *et al.*, 1989), (White, 1997). Although, 90% sensitivity has been reported in patients with more than two lesions; it declined to 50–70% with a single lesion (Del Brutto *et al.*, 1992) and as low as 28% has been found in cases with single cysts in the brain (Wilson *et al.*, 1991). EITB is an expensive, complex technique, requires expertise and moreover, facilities may not be available at various diagnostic laboratories or in field settings in the developing countries. This diagnostic technique has been previously discussed in the section dealing with porcine cysticercosis diagnostic tools but was first developed for use in humans, and not animals.

1.6.2.2 Ab ELISA

The Ab ELISA uses crude antigen for the detection of antibodies to *T. solium* in serum or cerebrospinal fluid (CSF). Antigens used in these tests are either cyst fluid or crude homogenates of *T. solium* cysticerci or crude preparations of the related parasite *T. crassiceps* , which can be maintained in laboratory rodents (Pardini *et al.*, 2002). These unpurified antigens have moderate sensitivities and relatively poor specificities (Schantz & Sarti, 1989), (Fleury *et al.*, 2001). In surveys on cysticercosis, antibody detection systems have been useful in identifying the risk factors associated with transmission of *Taenia solium* ; a high seroprevalence in a community indicates a ''hot spot'' where preventive and control measures should be applied (Dorny *et al.*, 2003). The screening for Ab ELISA used in this study was

done as described by (Guerra *et al.*, 1982). The antigen used for coating the polystyrene ELISA plates was a crude soluble extract of *T. solium* cysticerci.

The antigens used in the Ab ELISA, however, do react with antibodies from other helminthic infections, namely amoebas, filarisis and others as shown in the table below. These cross reactions limit the specificity of the Ab ELISA.

Table 3: Reaction of the blood of patients presenting with known heterological and homologous (*T. solium*) infections with the antigen *Taenia solium*

Known Infections	Number of sera samples tested	Number of sero-positive results (%)
Filaria 08 06 (75)	*8*	*6 (75)*
Strongyloides 05 02 (40)	*5*	*2 (40)*
Amoeba 05 02 (40)	*5*	*2 (40)*
Fasciola 05 03 (60)	*5*	*3 (60)*
Schistosoma 05 02 (40)	*5*	*2 (40)*
Echinococcus 06 06 (100)	*6*	*6 (100)*
C. cellulosae 07 07 (100)	*7*	*7 (100)*
Toxocara 08 02 (25)	*8*	*2 (25)*
Plasmodium 07 01 (14,28)	*7*	*1 (14)*

Source: Dorny, Pierre – personal communication

1.6.2.3 Ag ELISA

Originally this assay was developed for the detection of bovine cysticercosis and is based on a "sandwich" of 2 monoclonal antibodies raised against excretory/secretory products of *Taenia saginata* metacestodes (Brandt *et al.*, 1992; Dorny *et al.*, 2000). The adult tapeworm does not produce these antigens, and therefore, the test is very specific in detecting human *T. solium* cysticercosis with sensitivity and specificity of 92.3% and 98.7% respectively (Erhart *et al.*, 2002). The antigen ELISA seems to be a valuable tool in epidemiological studies of human cysticercosis. It may also be valuable as a confirmatory or initial test for individual diagnosis. Studies on bovine cysticercosis indicate that this test demonstrates infection with viable cysts only; this may also apply to human cysticercosis. The test may also be of use in the follow-up of neurocysticercosis patients. In Latin America, (Garcia *et al.*, 1998) and (Correa, 1999) also demonstrated the usefulness of serum antigen detection in cerebrospinal

fluid or serum for clinical diagnosis and monitoring of neurocysticercosis. Validation of the present monoclonal antibody-based antigen ELISA is in progress; preliminary results showed that serum samples from 34 of 36 cases of cysticercosis (94.4%), who had been confirmed by CT scan, biopsy of subcutaneous nodules, or both, gave positive results. Out of 41 patients with filariasis (three), amoebiasis (three), malaria (seven), schistosomiasis (three), trypanosomiasis (eight), hydatidosis (12) and cerebral tumors (five) only one cross-reaction was observed resulting in a specificity of 97.6%. Out of 84 documented cases of NCC, 74 (88.1%) were detected using the Ag-ELISA (Dorny et al. unpublished observation).

1.6.1.4 Neuroimaging techniques – Computed Tomography (CT) and Magnetic Resonance Imaging (MRI)

The diagnosis of neurocysticercosis has been greatly improved by the introduction of computed tomography (CT) and magnetic resonance imaging (MRI). These techniques demonstrate the number and topography of lesions, their stage of involution, and the degree of inflammatory reaction of the host against the parasites and have largely replaced previous radiological procedures such as plain roentgenograms, pneumoencephalograms, cerebral angiography and myelography. In general, MRI provides better image detection and definition. The possibility of multiplanar reconstruction of images, its capability to visualize the posterior fossa without bone artifacts, and its high contrast resolution (far superior to that of CT) allow MRI to recognize many forms of cysticercosis not visualized on CT. However, the costs of MRI are high and the equipment is scarcely available in many endemic countries, and its sensitivity for the detection of calcified lesions is poor. CT remains the best screening neuroimaging procedure for patients with suspected neurocysticercosis, and MRI is the imaging modality of choice for the evaluation of patients with intraventricular cysticercosis, brainstem cysts and small cysts located over the convexity of cerebral hemispheres. Its better image definition also suggests that MRI is superior to CT in the follow-up of the patients after therapy (Garcia & Del Brutto, 2003).

1.6.3 Treatment

There is no standard regimen for the treatment of neurocysticercosis but the treatment varies with the type of involvement and the presence of other factors. Management of neurocysticercosis includes the use of symptomatic medication (including anticonvulsants), anti-inflammatory drugs, anti-parasitic drugs, or surgery (Murrell 2005). The table below illustrates the criteria guiding clinical management of the disease.

Table 4: Treatment Management Guidelines for Neurocysticercosis

	Parenchymal neurocysticercosis
Viable (live cysts)	
One to five cysts	Anti-parasitic treatment, with corticosteroids
More than five cysts	Anti-parasitic treatment, with corticosteroids
	Anti-parasitic treatment, with high-dose corticosteroids. Alternatively, chronic steroid management. No anti-parasitic treatment, neuro-imaging follow-up
Enhancing lesions (degenerating cysts)	
Mild or moderate	No anti-parasitic treatment. Neuroimaging follow-up. Alternatively anti-parasitic treatment with corticosteroids and as a second alternative anti-parasitic treatment. Corticosteroids only if side effects develop
Heavy (cysticercotic encephalitis)	No anti-parasitic treatment. High dose corticosteroids and osmotic diuretics
Calcified cysticerci	
Any number	No anti-parasitic treatment
	Extraparenchymal neurocysticercosis
Ventricular cysticercosis	Neuroendoscopical removal, when available. If not available then CSF diversion followed by an anti-parasitic treatment with corticosteroids, and as a second alternative, open surgery (mainly for cysts in the fourth ventricle)
Subarachnoid cysts including giant cysts or "racemose" cysts on neuroimaging	Anti-parasitic treatment, with corticosteroids. Ventricular shunt if there is hydrocephalus
Hydrocephalus with no visible cysts on neuroimaging	Ventricular shunt. No anti-parasitic treatment
Other locations	
Spinal cysticercosis, intra or extramedullary	Primarily surgical. Anecdotal reports of successful use of albendazole with corticosteroids
Ophthalmic cysticercosis	Surgical resection of cysts

Source: (Murrell, 2005)

1.7 Risk factors associated with taeniasis and cysticercosis

Most of the risk factors associated with cysticercosis are associated with pig rearing and husbandry practices, hygiene and santitation practices and the consumption of pork. In addition, significance of risk has been associated with tapeworm infection in general and a tapeworm carrier in the household. Poverty is also associated with disease in general and the acquisition of helminthic infections. This chapter aims to establish risk factors for taeniasis/cysticercosis prevalence in order to identify those

with the most influence and to aid in diagnosis. The following sections of the introduction explore the literature to put factors that are investigated in this chapter into context. The factors under consideration will in some cases be associated with both human taeniasis/cysticercosis and porcine cysticercosis. Factors to be explored are: hygiene and sanitation practices (source and treatment of household water, presence and use of latrine, ingestion and preparation of pork), household level indicators (age, education level, presence of tapeworm carrier in household), factors associated with pig keeping (occupation, grazing practices, types of feed, inspection of carcass after slaughter), and knowledge of tapeworm (nodules observed in humans and pigs, observation of passing of proglottids).

1.7.1 Hygiene and Sanitation Practices

1.7.1.1 Source and Treatment of Water

The source and treatment of water used in the household have been identified as risk factors in the transmission of cysticercosis. Cysts in humans develop at the end of a cycle which previously relies on the ingestion of ova of *Taenia solium* which are excreted by human carriers in their feces. The risk of contamination with *Taenia* ova is related to the contact with *Taenia solium* carriers. The most common means of transmission, however, is ingestion of *Taenia solium* eggs from contaminated food or water (Antoniuk, 1999). Water that is collected from a source such as a river or uncovered well could easily be contaminated with faeces and thus contain eggs. Studies from Central and Latin America have identified drinking unboiled water and not properly washing hands before and after eating as risk factors for cysticercosis (Garcia *et al.*, 1995), (Sarti *et al.*, 1996). In a community-based study in Shandong, China, drinking unclean water, not washing hands after defecation and eating food with dirty hands indicated a two-fold greater odds of cysticercosis morbidity, the findings of which indicated an overall prevalence of 3.2% (Cao *et al.*, 1996).

1.7.1.2 Latrine Use and Presence and Defecation Habits

Not using a latrine when defecating and defecation in pigstys have a significant effect on cysticercosis prevalence in humans (Cao *et al.*, 1996). If pigs are allowed to ingest human faeces contaminated with the *Taenia solium* tapeworm, they acquire

porcine cysticercosis and the cycle of transmission is perpetuated. In a study of prevalence and risk factors for taeniasis and cysticercosis in humans and pigs in Mexico, infection rates of porcine cysticercosis increased with the age of pigs, and were habitually higher in pigs that had access to latrines or were fed human faeces (Sarti *et al.*, 1992). In another study which examined the epidemiology and prevalence of *Taenia solium* taeniasis and cysticercosis in Guatemala, however, no significant relationship between seropositivity and risk factors such as water supply and the absence/presence or type of toilet was observed. The results of this immunoblot study indicated a prevalence of 17.3% in one community and 10.1% in the other. It was observed that the community that recorded the higher rate of seropositivity was the community which was consistently poor socieconomically, with lower adult literacy rates, more households defecating in the open, having no means of waste water removal from the home (waste water generally poured on to the ground around the house) and fewer houses with piped water (Garcia-Noval *et al.*, 1996). Similarly, a study in Mbulu District, Tanzania, also reported no significant association between the absence of latrines and the prevalence of infection (OR = 0.23; 95% CI = 0.06, 0.91) (Ngowi *et al.*, 2004). This difference in significance of risk factor can also largely be dependent on the condition of the latrine if present. If the latrine is present and clearly in use, but does not have barriers to restrict the access of pigs, having a latrine may in fact increase the odds of pig cysticcerosis.

1.7.1.3 Ingestion of and Preparation of Pork

Eating raw or inadequately cooked pork is strongly associated with the risk of acquiring taeniasis caused by *Taenia solium*. The intestinal tapeworm infection is acquired when larval forms (cysticerci) are ingested in raw or insufficiently cooked pork. Studies from Mexico and Peru have identified the consumption of uncooked or infected pork as being strongly related to seropositivity for taeniasis (Sarti *et al.*, 1992; Garcia *et al.*, 1995). In another study in Mexico City which examined the prevalence and risk of cysticercosis and taeniasis amongst and urban population of soldiers and their families, relatives of the soldiers positive for cysticercosis and taeniasis markers ate more pork from street vendors than restaurants or markets compared with relatives of soldiers without these indicators of infection (Garcia-

Garcia *et al.*, 1999). Pork that is prepared in an informal setting has a greater chance of being poorly cooked or has potentially never been inspected for disease owing to the lack of enforcement of food hygiene regulations and may therefore contain cysticerci which are then consumed. In a study in Zambia, the majority of farmers (92.3%) consumed pork and the prevalence rate for porcine cysticercosis showing seropositivity by Ag ELISA was 37%. Such a high rate of porcine infection and pork consumption suggests the human infection rate might be high as well (Sikasunge *et al.*, 2007).

1.7.2 Indicators at Household Level

1.7.2.1 Age

The age of human cysticercosis seropostiive subjects has generally had an association with seropositivity in studies of prevalence and risk factors (Diaz *et al.*, 1992a; Sarti *et al.*, 1994; García *et al.*, 1997). In a study of pig and human cysticercosis in Nigeria, in which the overall prevalence of taeniid ova in the 1525 human-stool samples analysed was 8.6%, most (78.6%) of the cases occurred in adults aged > 30 years (Onah & Chiejina, 1995). In a study or rural households in Bolivia, seroprevalence increased gradually with age, with a high of 44% for ages 51–60 (Carrique-Mas *et al.*, 2001). This can be explained by the fact that the longer people are exposed to *T. solium* eggs in an endemic environment, the more chances they have to become infected (Zoli *et al.*, 2002). Conversely, in a study of 317 villagers in Peru, in which *Taenia solium* specific antibodies were detected in 21% of the sample, although the frequencies of seropositivity were similar for males and females, (24% and 18% respectively) the subjects aged 4-29 years were more likely to be seropositive than the older subjects ($P<0.001$). A positive result in the EITB assay was significantly associated with being aged <30 years in both the univariate and logistic regression analysis (Moro *et al.*, 2003). This unusual distribution may be explained by an early, intense exposure to *T. solium* eggs in a heavily contaminated environment or that some of the positives to the EITB were in fact false positives to *T. solium* and the subjects contained antibodies against *Echinococcus granulosus*. This latter theory, however, would call into question the specificity of the EITB, which has consistently been reported to be 100% (Tsang *et al.*, 1989; Wilson *et al.*,

1991). The association between younger age groups and increased prevalence of active cysticercosis was also found in a hyperendemic area of Venezuela, a study in which Ferrer *et al.* (2003) did find more cases in individuals aged ≤ 30 years. The explanation for this relative anomaly was that in older individuals cysts can start to die and calcify, resulting in late onset epilepsy and the Ag ELISA would therefore not have detected viable cysts in older age groups, merely the presence of live cysts in younger patients (Ferrer *et al.*, 2003).

1.7.2.2 Education

The level of education in subjects has been shown to have an association with prevalence rates of taeniais in studies from Central and Latin America. In a study carried out in Peru, results from a multiple logistic regression analysis for best fit with a positive electroimmunoblot transfer result (EITB) as outcome showed an almost three fold greater odds of being positive when the subject possessed an education level of no more than elementary level (Moro *et al.*, 2003). In a study in the Bolivian Chaco, seroprevalence was negatively associated with level of education, the data being analysed for people over 26-years-old only: it was 16/39 (41%) in illiterates, 25/90 (27.8%) in those with primary education only, 7/34 (20.6%) for secondary educated and 3/19 (15.8%) for university educated people (Carrique-Mas *et al.*, 2001). There are, however, several studies in which there is no statistical significance between education levels and prevlance of *Taenia solium* cysticercosis (Garcia *et al.*, 1995; Cao, 1997; Goodman *et al.*, 1999). In the case control study by Goodman et al., in which both the cases and controls had similarly high levels of education (17% and 20% respectively high school or above), 12 % of the cases and 4 % of the controls were seropositive by EITB, indicating that a high level of education does not necessarily correlate negatively with a high prevalence.

1.7.2.3 Presence of tapeworm carrier in household

Data from the Americas indicates that the main risk for both human neurocysticercosis and swine cysticercosis is the presence of a tapeworm carrier in the household (Flisser *et al.*, 2003). In 1988, a survey performed in the community of El Sotano in Mexico showed for the first time the association of the parasite's life

cycle with pigs, humans and tapeworms, as people with a history of passing proglottids or with *Taenia* eggs in their stools were detected in the same houses with people that had anti-cysticercus antibodies and with pigs that had cysticercosis (Sarti *et al.*, 1988). Another study reported the finding of carriers of the adult *T. solium* who had significant numbers of people with anti-cysticercus antibodies living in close proximity (Diaz-Camacho *et al.*, 1990), thus reinforcing the close association of the life cycle – tapeworm carriers, pigs with cysticercosis and seropositive people all occurring close to each other. Other studies from Bolivia, Ecuador and Guatemala indicate this same association (Cruz *et al.*, 1989; Garcia-Noval *et al.*, 1996; Bern *et al.*, 1999). In a study of the rural population in Peru, (Garcia *et al.*, 2003b)) found 0-6.7% (median 2.5%) taeniasis cases by copro-antigen ELISA, while the seroprevalence for cysticercosis by EITB in the same population varied from 7.1 to 26.9% and persons living with tapeworm carriers had 4.3 times greater odds of testing positive than those living without a tapeworm carrier. Further corroboration of the increased risk of locally acquired cysticercosis is borne out by a study conducted to determine the potential for infection from immigrant domestic workers of an orthodox Jewish population in New York City. Adherence to dietary laws that prohibit the ingestion of pork is the norm of this population thereby implying maintenance of the life cycle of *Taenia solium* solely within members of this community is not possible. Almost all (99.6%) domestic employees were immigrants and 97% were from Latin America. For housekeepers employed in the last five years, a Central American country of origin was associated with seropositive households (RR=2.7, P=0.0001). Of the 1,789 members of the community studied, 23 (1.3%) were seropositive by EITB assay. Being female was also associated with seropostivity which reinforces the influence of the domestic worker as a risk factor. In this community females do not traditionally work outside the home so would have the most potential for contact with the domestic worker. Somewhat surprisingly, children of less than five years old had a significantly lower EITB seroprevalence than other age groups but a greater frequency of contact with the infected domestic worker. This, however, could reflect the shorter time at risk for exposure as children aged 6-10 years had a seroprevalence of 1.7% (Schantz *et al.*, 1992; Moore *et al.*, 1995).

1.7.3 Factors associated with Pig-keeping

1.7.3.1 Raising Pigs

In a study of epileptic patients in Leon, Nicaragua, the only statistically significant association for seropositivity for taeniasis/cysticercosis by EITB or ELISA was the subject living in a household in which pigs were raised (OR = 5.18, CI = 0.8-41.6; $P < 0.05$) (Bucardo *et al.*, 2005). This significance is borne out by several other studies conducted around the world. In China, in a population-based case-control study of rural respondents in Shandong Province, those who raised pigs had an almost 3 fold greater association with seropositivity, a relationship which was statistically significant (OR = 2.6, CI = 1.2-5.7; $P < 0.05$) (Cao, 1997). In Peru, raising pigs, irrespective of the number, was associated with being seropositive (232/1,562, 14.9% versus 123/1,021, 12.0%; OR 1.27; CI 1.01, 1.61; $P < 0.043$) (Garcia *et al.*, 1995; Garcia *et al.*, 2003b). In India, where it was found that 78% of children of pig farmers passed taeniid eggs in their stools (Banerjee *et al.*, 1994), having no separate place to keep pigs apart from the household space and having epilepsy in the family were identified as risk factors for NCC clustering in a family (Prasad *et al.*, 2009).

1.7.3.2 Pig Husbandry Practices

Allowing pigs access to human faeces is an independent risk factor for cysticercosis morbidity, whereas raising pigs is independently associated with seropositivity, according to a study carried out in China (Cao *et al.*, 1996; Cao, 1997). In the study area, pigs are raised in households and most are allowed to graze on human faeces; this can be responsible not only for a high rate of porcine infection, but also favours the transmission of human cysticercosis within the community as pigs can help spread the parasite eggs in the environment. In a study conducted in Mbulu District in Tanzania, 96% of pigs tested for cysticercosis ranged free. The prevalence rate obtained for this study was 17% by lingual examination, a method which has lower accuracy than serology (Ngowi *et al.*, 2004). Husbandry practices such as tethering some of the time or fencing the pigs separately from the household have been shown to have a negative effect on serological association as pigs do not have access to

faeces, which can potentially break the life cycle of the parasite (Cao, 1997). Free-ranging pigs can be fed at minimal cost since the majority of their food is obtained by scavenging. However, corralling pigs, thus restricting their access to human feces, although more expensive in the short term, can be promoted as cost-effective because a cyst-free pig is generally sold at much higher prices than an infected one. Since sanitary conditions (latrines, sewage disposal, and potable water) are difficult to achieve and unlikely to be generally improved in rural communities of developing countries, control of cysticercosis through sound pig husbandry practices appears to be the most accessible and cost-effective target for control programs (Garcia *et al.*, 1995).

One of the main indicators of success in controlling cysticercosis is environmental contamination as measured by the level of swine cysticercosis (Gonzalez *et al.*, 1994). A study performed in the community of Coapeche in Veracruz showed that none of the pigs (53) examined by tongue palpation had cysticercosis and no antibodies were detected by western blot. The survey showed that all pigs were restrained and that 91% of houses had latrines (Flisser *et al.*, 2003). Economics can also play a motivating factor in control of pigs as people have learnt that if pigs are restrained and have no access to faeces, the meat can be sold at a higher price as it is uncontaminated (Sarti *et al.*, 1997b).

1.7.3.3 Meat Inspection

Lack of inspection of pork before and after slaughter contributes to the continuing spread of cysticercosis and management of such can be looked at as an entry point for control of the disease (Ngowi *et al.*, 2004; Boa *et al.*, 2006). In many areas of Dar es Salaam in Tanzania, slaughtering pigs is almost an exclusively backyard operation (Airey, 1995), and many pig keepers sell pork directly to their neighbours and friends on a pre-order basis. Lack of slaughter facilities and meat inspection in many areas of the country is a threat to food safely and guidelines for inspection of pork for porcine cysticercosis are not present within the national meat inspection regulations of Tanzania, although provisions have been made for bovine cysticercosis (Boa *et al.*, 2006). A recent study conducted in Mbulu District found that detection of porcine

cysticercosis in slaughter pigs is low when the current pork inspection regulations are followed (Boa *et al.*, 2002). In Zambia, pigs are mainly raised by resource-limited farmers. In the Eastern province, pigs are raised mainly for home consumption, while most of the pigs raised in the Southern province are transported to Lusaka where they are sold and slaughtered at an illegal slaughter slab. Recent (unpublished) observations showed a high prevalence of *T. solium* cysticercosis in pigs presented at this slaughter slab (Phiri, 2002).

1.7.4 Knowledge of Tapeworm

1.7.4.1 Nodules observed under skin (pigs or humans)

There is a strong association with nodules observed under the skin and positive serology for human cysticercosis. In a study in Irian Jaya, Indonesia, of the 31 patients reporting to a local health centre with epilepsy and subcutaneous nodules, Immunoblot analysis revealed that 12 (67%) of 18 and 20 (65%) of 31 cases, respectively, were serologically confirmed as cysticercosis (Wandra *et al.*, 2000). The failure of people to recognize the presence of cysts in pigs as being positively associated with cysticercosis morbidity contributes to the lack of control of the disease. In a study in Zambia, observation of cysts by farmers was high in all districts, but did not deter them from eating or selling pork. Most of the respondents (83.3%) had observed cysts in pork. Of the respondents that observed cysts in pork, 20.1% ate and 18.3% sold the meat. Of the 659 farmers who observed cysticercosis, 43.8% acknowledged pork measles as just being a pig disease, 6.5% referred to it as being husks or maize bran (because they think that porcine cysticercosis results from feeding pigs with maize bran or husks) whereas, 48.3% had no idea of what cysts seen in pork was indicative (Sikasunge *et al.*, 2007).

1.7.4.2 Awareness of disease or tapeworm

In a health awareness intervention study carried out in Mexico designed to educate the community on the life cycle of the parasite, the diseases and control measures regarding cysticercosis, (Sarti *et al.*, 1997b) found that community awareness of the disease can play an important role in control of porcine cysticercosis. The prevalences of cysticercosis in pigs at the start of the education intervention were

2.6% and 5.2% by lingual examination and antibody detection (immunoblot assay), respectively, and approximately one year after the intervention they were 0% and 1.2% ($P < 0.05$). These changes were accompanied by significant reductions in the reported access of pigs to sources of infection and freedom to roam. The study concluded that health education, developed along with community involvement, reduced opportunities for transmission of *T. solium* in the human-pig cycle (Sarti *et al.*, 1997a). In a study conducted by (Ferrer *et al.*, 2003), paradoxically, higher seroprevalences were recorded in the two communities that were most aware, and informed of the risks presented by Taenia solium. Evidence of taeniasis, which was indicated by passing of tapeworm proglottids, was also higher in these two communities. Although sanitary education had been carried out, behavioural change had not necessarily followed. The habit of eating potentially infected pork and the lack of sanitary facilities or latrines presented ideal conditions for porcine parasite transmission. In this case, sanitary education alone was not sufficient to control the disease.

Individuals without knowledge on infected meat have a higher risk of getting taeniasis and may subsequently get cysticercosis. In a study in Shandong Province, China, this risk factor is significant for seropositivity as well as cysticercosis morbidity (OR = 11.7, CI = 1.9 - +INF; $P < 0.01$). This lack of awareness may continue to play a role in the future as with the establishment of a freer market economy in China, many people have gone into business selling pork on the local market and at the same time regulations for inspection of meat have become less stringent. Presently meat can be sold without official examination, allowing cysticerci-containing pork to readily be obtained (Cao, 1997). The link between lack of knowledge of association between infected meat in pigs and cysticercosis in humans is further reinforced by data from a study in Zambia. In this study, 85.5% of the farmers in the Eastern province and 98.0% in the Southern province did not know that there exists a link between human taeniasis and porcine cysticercosis. Of the farmers interviewed, 87.3% in the Eastern province and 98.5% in the Southern province did not know that tapeworm infection in humans was due to eating infected pork. Ninety-seven percent of the farmers did not know that pig cysticercosis was

due to pigs eating human faeces contaminated with *T. solium* eggs (Sikasunge *et al.*, 2007).

1.7.4.3 History of passing proglottids

In studies from Central America, a history of passing proglottids or tapeworm sections in faeces is significantly associated with seropositivity to an EITB for human cysticercosis (Sarti *et al.*, 1992; Ferrer *et al.*, 2003). In a survey of 1,552 persons queried during a household survey, 90 (5.8%) reported a history of passing tapeworm proglottid in faeces (this question was further explained by showing the respondents pictures and fixed specimens of proglottids). The principal risk factors associated with a history of passing proglottids in faeces were having owned infected pigs (OR = 2.65, CI = 1.37-5.12; $P < 0.004$) and having eaten infected pork (OR = 1.93, CI = 1.0-3.76; $P < 0.05$). Goodness of fit analysis confirmed that seropositive persons (but not infected pigs) were significantly clustered within households, particularly in households in which a member reported a history of have passed proglottids ($x^2 = 31.95$, $P < 0.005$). In a study examining neurocysticercosis in endemic villages in Peru, a significantly high proportion of those tested stated a history of passing proglottids. Of those with a positive EITB (Tsang *et al.*, 1989), 47.8% (11/23) reported having passed proglottids (García *et al.*, 1997). It must be noted, however, that in this study there was a lack of specificity when asking the question regarding passing of proglottids and no visual tools were used, therefore allowing for some degree of error or confusion with the answers. Nonetheless, since there was a significant association between such a history and a positive EITB result, autoinfection may be important in the acquisition of neurocysticercosis within the population (García *et al.*, 1997). Similarly, in a study carried out in a rural pig farming community in India, factors associated with taeniasis on the multivariate analysis were above age 15 yrs, history of passing *Taenia solium* segments in stool (OR = 3.31, CI = 2.31-4.76; $P < 0.001$), undercooked pork consumption and poor hygiene in hand washing (Prasad *et al.*, 2007). The presence of *Taenia* carriers living within the community is a potential threat as is borne out by literature on the previously described risk associated with taeniasis/human cysticercosis and living in

close proximity to a tapeworm carrier (either within the household or in close association) (Garcia *et al.*, 2003b).

1.8 Epilepsy

1.8.1 General Background

The word "epilepsy" derives from the Greek verb "epilambanein", meaning "to be seized, to be overwhelmed by surprise" and captures well the sudden, usually unpredictable and intrusive nature of most seizures. Neurologists define epilepsy as: "a condition in which individuals experience paroxysmal changes in behaviour caused by abnormalities in the electrical activity of the brain" (Asbury, 1992). In other words, epilepsy is the name given to a group of functional disorders of the brain that are characterized by repetitive seizures. Seizures involve abnormal, excessive electric discharges of groups or assemblies of nerve cells (neurones) in the brain (WHO, 2004a).

Epilepsy is the most common serious neurological disorder and is one of the most prevalent non-communicable disease in the world (Scott, 2001). The WHO Neurosciences Research Protocol for studying the prevalence of neurological disorders in developing countries defines epilepsy as two or more afebrile seizures unrelated to acute metabolic disorders or to withdrawal of drugs or alcohol (Senanayake, 1993). According to the WHO "Out of the Shadows" – Global Campaign Against Epilepsy, in all areas of the world, no less than three out of every thousand people – and in several places over 40 per thousand (4%) are affected.

Approximately 2.5 million people worldwide carry adult *T. solium* (Burneo, 2001). The prevalence of epilepsy in industrialized countries is about 3-9 per 1000 population. The prevalence in developing countries, data which is based largely on community surveys of rural populations, is as high as 49 per 1000 population in Liberia and 57 per 1000 in Panama (Senanayake, 1993).

1.8.2 Prevalence in Sub Saharan Africa

The prevalence of epilepsy for sub-Saharan Africa varies considerably, with smaller studies showing in general a higher prevalence and larger studies showing prevalence nearer that in the developed world. The median prevalence found using door-to-door studies is 15 per 1000 people (Preux, 2005). The table below illustrates varying rates of prevalence using door-to-door studies in sub-Saharan Africa.

Table 5: Prevalence of Epilepsy in sub-Saharan Africa using door-to-door surveys

Country	Reference	Year	N	Prevalence (per 1000)	95 % CI
Benin (Agbogbomé)	(Gbenou, 1995)	1995	530	24.5	10.9-38.1
Benin (Savalou)	(Avodé, 1996)	1996	1,443	15.2	8.7-21.7
Benin (Zinvié)	(DeBrock, 2000)	2000	3,134	33.5	22.3-44.3
Burkina Faso	Debouverie, 1993)	1993	16,627	10.6	9.1-12.2
Cameroon	(Nkwi, 1989)	1989	500	70.0	46.3-93.6
Cameroon (Bilomo)	(Dongmo, 2000)	2000	1,900	58.4	46.9-69.1
Ethiopia (Butajira)	(Tekle-Haimanot, 1990)	1990	60,820	5.2	4.6-5.8
Ivory Coast	(Kouassi, 1998)	1988	1,176	7.6	2.5-12.7
Ivory Coast	(Kouadjo, 1990)	1990	309	74.4	43.0-104.9
Ivory Coast (M'Brou)	(Kaudjhuis, 1995)	1995	920	59.0	43.0-75.0
Kenya (Nakuru)	(Kaamugisha & Feksi, 1998)	1988	2,960	18.2	13.2-23.2
Liberia	(Goudsmit, 1983)	1983	4,436	28.0	23.0-33.0
Madagascar	(Andriantseheno, 2004)	2001	925	20.8	11.3-30.3
Mali	(Farnarier, 2000)	2000	5,243	15.6	12.2-19.0
Mali (Bamako)	(Traoré, 2000)	2000	4,074	11.3	8.0-14.6
Nigeria	(Longe, 1989)	1989	2,925	6.2	3.3-9.1
Nigeria (Aiyété)	(Osuntokun, 1982)	1982	903	37.0	24.2-49.8
Nigeria (Igbo-Ora)	(Osuntokun, 1987)	1987	18,954	5.3	4.2-6.4
Senegal	(Ndiaye, 1986)	1986	7,682	8.3	6.2-10.4
Senegal	(Diop, 1996)	1996	2,803	21.0	15.5-26.5
Tanzania	(Rwiza, 1992)	1992	18,183	10.2	8.7-11.7
Togo (Akebou)	(Grunitzky, 1996)	1996	4,182	13.1	9.6-16.6
Togo (Kloto)	(Grunitzky, 1991)	1991	19,241	12.3	10.7-13.9
Togo (Kozah)	(Dumas, 1989)	1989	5,264	16.7	13.1-20.3
Togo (Tone)	(Balagou, 2000)	2000	9,143	18.6	15.7-21.5
Uganda	(Kaiser, 1996)	1996	4,743	13.0	9.7-16.3
Zambia (Chikankata)	(Birbeck, 2004)	2004	55,000	12.5	11.5-13.5

Source: (Preux, 2005)

It is estimated that 10% of the burden of brain and mental disorders in the world is caused by epilepsy, calculated in disability-adjusted life years (DALYs), which is very significant. This calculation includes premature deaths and the loss of healthy life due to disability (World Bank, 1993). Every year, among every 100,000 persons, there will be 40-70 new cases (WHO, 2003c). Of the 50 million people in the world who have epilepsy, approximately 35 million have no access to appropriate treatment. This is in most cases because services are unaffordable or non-existent and in some cases because epilepsy is not viewed as a medical problem or as a treatable brain disorder (WHO, 2001b).

1.8.3 Burden of Disease Estimates for Epilepsy

The burden of disease (BOD) estimates for epilepsy include epilepsy and status epilepticus. (Mathers, 2003) estimate the DALYs for epilepsy as 6,223,000, with slightly higher rates for males (3,301,000) than for females (2,922,000). Many risk factors for epilepsy are linked with a lower level of economic development; thus, the burden is highest in South Asia followed by Sub-Saharan Africa (Table 4). A notable observation is the reportedly low burden in the Middle East and North Africa, despite parts of that region being relatively underdeveloped. Epilepsy imposes a large economic burden on patients and their families. It also imposes a hidden burden associated with stigmatization and discrimination against patients and even their families in the community, workplace, school, and home. Social isolation, emotional distress, dependence on family, poor employment opportunities, and personal injury add to the suffering of people with epilepsy (Jamison, 2006).

Table 6: Disability-Adjusted Life Years by Cause and Region, 2001 (x1000)

Condition	Global total			East Asia & Pacific	Europe & Central Asia	Latin America & the Caribbean	Middle East & North Africa	South Asia	Sub-Saharan Africa Countries	High-income Countries
	Mixed sexes	Males	Females							
AD and other Dementias	17,108	6,092	11,016	4,110	1,612	1,215	292	1,955	450	7,468
Epilepsy	6,223	3,301	2,922	1,303	354	737	248	1,741	1,373	464
PD	2,325	1,124	1,202	435	228	90	81	303	100	1,086
Cerebrovascular Disease	72,024	35,482	36,542	25,832	12,616	3,936	1,948	13,184	5,125	9,354

Source: (Mathers, 2003)

More than 80% of patients with epilepsy live in developing countries and most of these live in sub-Saharan Africa (WHO, 2001b). Data on the incidence of and prognosis for epilepsy in sub-Saharan Africa are scarce but prevalence data show that epilepsy is two to three times more common than in industrialized countries in non-tropical areas (Preux, 2005).

1.8.4 Definitions for Epilepsy Diagnosis

Epileptic seizure. A clinical manifestation presumed to result from an abnormal and excessive discharge of a set of neurons in the brain. The clinical manifestation consists of sudden and transitory abnormal phenomena which may include alterations of consciousness, motor, sensory, autonomic, or psychic events, perceived by the patient or an observer.

Epilepsy. A condition characterized by recurrent (two or more) epileptic seizures, unprovoked by any immediate identified cause. Multiple seizures occurring in a 24-h period are considered a single event. An episode of status epilepticus is considered a single event. Individuals who have had only febrile seizures or only neonatal seizures as herein defined are excluded from this category.

Status epilepticus. A single epileptic seizure of >30-min duration or a series of epileptic seizures during which function is not regained between ictal events in a >30-min period.

"Active" epilepsy. A prevalent case of active epilepsy is defined as a person with epilepsy who has had at least one epileptic seizure in the previous 5years, regardless of antiepileptic drug (AED) treatment. A case under treatment is someone with the correct diagnosis of epilepsy receiving (or having received) AEDs on prevalence day.

Epilepsy in remission with treatment. **A** prevalent case of epilepsy with no seizures for ≥ **5** years and receiving AED at the time of ascertainment.

Epilepsy in remission without treatment. A prevalent case of epilepsy with no seizures for ≥5 years and not receiving AED at the time of ascertainment.

Single or isolated seizure. One or more epilepticseizures occurring in a 24-h period.

Febrile seizure. An epileptic seizure as herein defined, occurring in childhood after age 1 month, associated with a febrile illness not caused by an infection of the CNS, without previous neonatal seizures or a previous unprovoked seizure, and not meeting criteria for other acute symptomatic seizures.

Neonatal seizure. An epileptic seizure as herein defined occurring in the first **4** weeks of life.

Febrile seizure with neonatal seizure. One or moreneonatal seizures in a child who has also experienced one or more febrile seizures as herein defined.

Nonepileptic events. Clinical manifestations presumed to be unrelated to an abnormal and excessive discharge of a set of neurons of the brain, including:

- disturbances in brain function (vertigo or dizziness, syncope, sleep and movement disorders, transient global amnesia, migraine, enuresis), and pseudoseizures (nonepileptic sudden behavioral episodes presumed to be of psychogenic origin; these may coexist with true epileptic seizures).

All definitions have been taken from the Commission on Classification and Terminology of the International League Against Epilepsy (ILAE, 1989).

1.8.5 Treatment of Epilepsy

A large proportion of the 50 million people affected by epilepsy remains untreated (Ellison *et al.*, 1988). In India, for example, of the 10 million people thought to have epilepsy, 5 million of them remain untreated (Gourie-Devi *et al.*, 1999). This treatment gap has been measured by the International League against Epilepsy as the difference between the number of people with active epilepsy (two or more unprovoked seizures on different days in the previous year) and the number whose seizures are being appropriately treated in a given population at a given point in time, expressed as a percentage (Meinardi *et al.*, 2001). It should also be noted that while four-fifths of the potential market for antiepileptic drugs is in the developing world, up to 90% of people with epilepsy in developing countries receive no treatment at all (Shorvon & Farmer, 1988), (Kale, 1997).

In a review of pharmacological treatment therapy in Africa, the ILAE found that In French-speaking areas, phenobarbitone is prescribed in 65–85% of treated cases. In English-speaking countries, phenytoin (PHT) is also frequently prescribed. Carbamazepine (CBZ) is the second drug, but is prescribed in only 5–20% of treated cases. Valproate (VPA) is prescribed in 5–15%, but is less widely available, and costs are also much higher than those of the aforementioned drugs. For status epilepticus, injectable PB or diazepam (DZP) is used when available (Meinardi *et al.*, 2001).

A study conducted in rural and semi-rural areas around Nakuru, Kenya, looked at (amongst other indicators), the clinical effectiveness of a treatment programme in terms of seizure control, toxicity and compliance and whether control of generalized tonic-clonic seizures inthis group is more difficult when started late. The study put patients on a regimen of drug therapy of carbamazepine or phenobarbitone, the patient and drug having been selected at random. The results of the study included

the findings that either drug was effective in reducing seizure frequency ($P < 0.05$) and there was no difference in the effectiveness of treatment with respect to the length of history of epilepsy and the lifetime number of seizures (Feksi *et al.*, 1991).

1.8.6 Social Effects of Epilepsy

The hypothesis generally is that epilepsy is a disease incurring considerable social disadvantage, not to say stigma, a hypothesis borne out in the experience of most researchers (Shorvon & Farmer, 1988). It is also a disease about which many sufferers are reluctant to discuss. Research in Nigeria which concentrated on obtaining evidence from the patients themselves on the social disadvantages of the disease, found that this information was not forthcoming since sufferers are likely to be reluctant to admit to such sensitive problems.

Epileptics often face problems of stigmatization from their own communities. In Ethiopia, patients were treated as lepers and banished from the community (Geil, 1968). Patients in native American communities in the United States have been accused committing incest, an offence which has resulted in acquiring the disease (Levy *et al.*, 1979). In Madagascar, epileptic patients were buried separately (Osuntokun, 1977). In Nigeria, nonepileptic respondents asked to compare patients cured of psychosis with epileptic ones discriminated on the whole in favour of the ex-psychotics (Arawatife *et al.*, 1985). Questionnaires administered in Kenya and Ecuador to nonepileptic controls and community leaders revealed some realistic assessment of epilepsy both as a disease and a social condition, but also much prejudice. Informants claimed they would not let their children play with known epileptics, would not marry them, or let their children do so. Epileptic patients are thought to be uneducable, unemployable, a danger to the community. The inability to get married, breakups of marriages and problems with employment seem factors in the lives of epileptic sufferers almost everywhere, but by no means invariably (Shorvon & Farmer, 1988).

1.9 Summary of Research Objectives

The aim of this study is to find the prevalence of NCC-related epilepsy in a group of previously identified epilepsy sufferers in Busia District, Western Kenya. The study will assess known risk factors for taeniasis and NCC which have been discussed in this chapter, using a questionnaire and administer serological testing within the study population, using tests known to be specific and sensitive for the presence of *Taenia solium* in humans. The study attempted to find a correlation between the presence of these risk factors and a positive test result.

Chapter 2 - Description of Study Area

2.1 *Introduction*

This chapter aims to describe the study area, namely Busia District. As the District had reported increases in pig – keeping in recent years (Thuranira, 2005), it was possible that with this increase would have accompanied a potential increase in the incidence of neurocysticercosis. Participants for the study were selected on the basis of an epilepsy diagnosis by a qualified professional and were taken from all areas of the District. A brief description of the District with its Divisions and Locations follows.

2.2 *Study Area General Description*

2.2.1 Location and Administration

Busia District is located in Western Kenya, bordering Uganda on the west side, Teso District to the north, Siaya and Mumias Districts to the east and Lake Victoria to the south. Busia District falls within the Lake Victoria Basin.

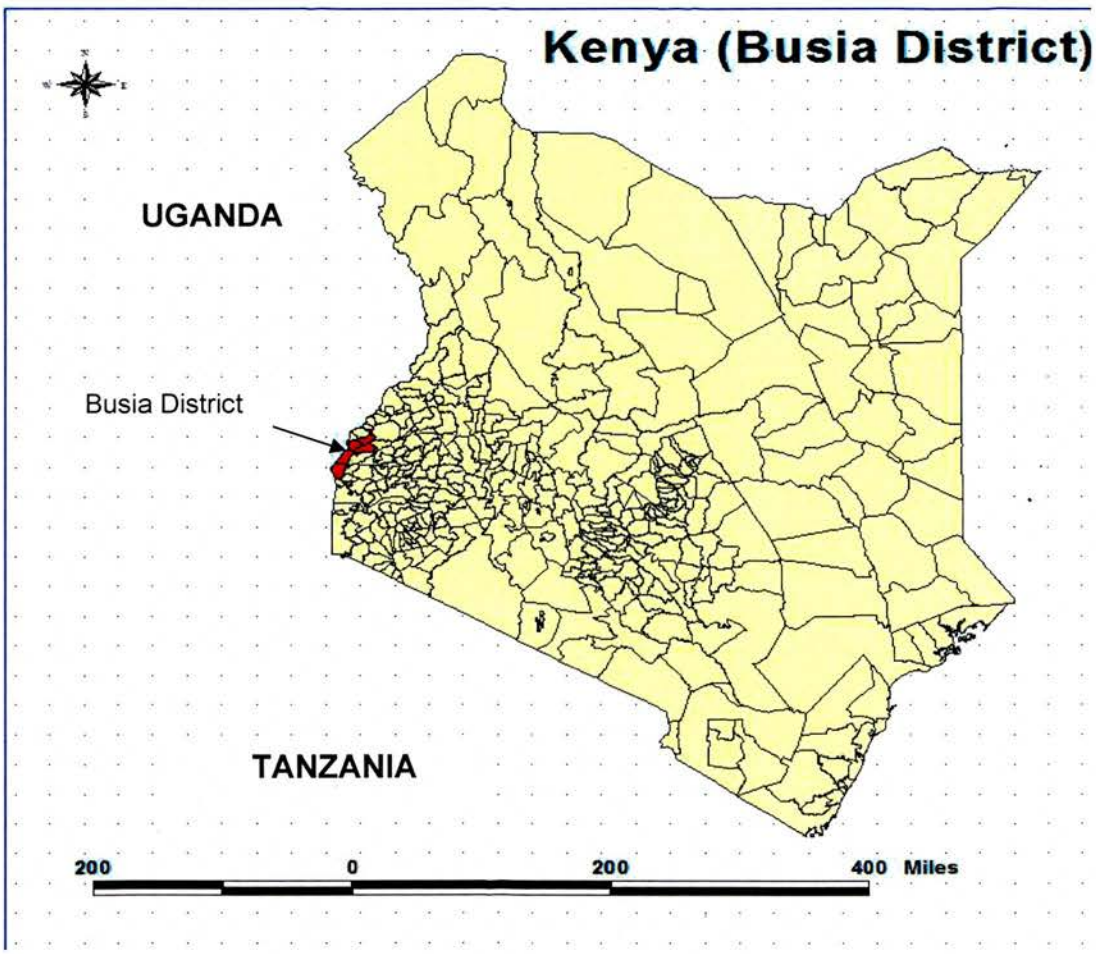
The altitude varies from 1,130 m above sea level on the shores of Lake Victoria to 1 375 m above sea level in the central part. The northern divisions (Butula and Nambale) occupy a plain characterised by low flat divides. These are often capped by laterites and shallow incised swampy systems. The peneplain has fertile soils suitable for growing maize, Robusta coffee and sugar cane.

The southern part, which covers parts of Matayos Division, Funyula Division and the northern part of Budalang'i Division is covered by a range of hills comprising the Samia Hills, which run from northeast to southwest, culminating at Port Victoria. The Yala Swamp is found in the extreme south of the district, an area which forms a colony of papyrus growth broken by irregular water channels and occasional small lakes with grassy islands (CBS, 2000; Government of Kenya, 2002).

The district is divided into six divisions: Funyula, Butula, Nambale, Matayos, Budalang'i and Busia Municipal or Town. These are further divided into 30 locations

and 99 sub-locations with individual villages within these sub-locations being the smallest administrative units. This study involved participants from all divisions.

Figure 5: Map of Kenya showing Busia District



The following maps show Busia District divided into Divisions (Figure 6) and the locations of the study subjects (Figure 7).

Figure 6: Map of Busia District with Divisions

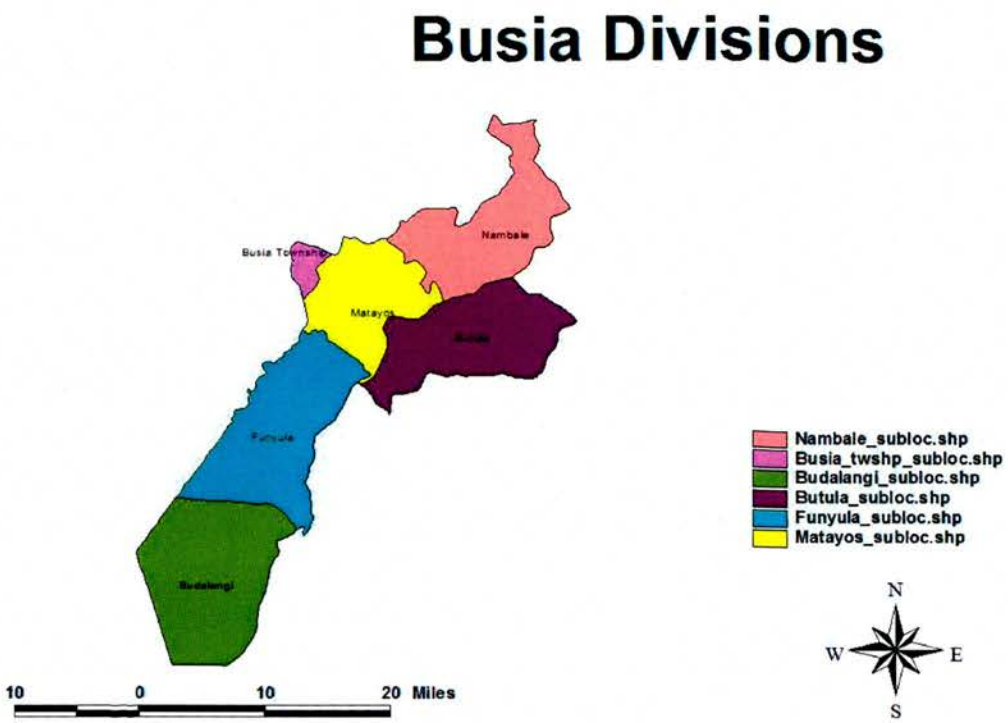
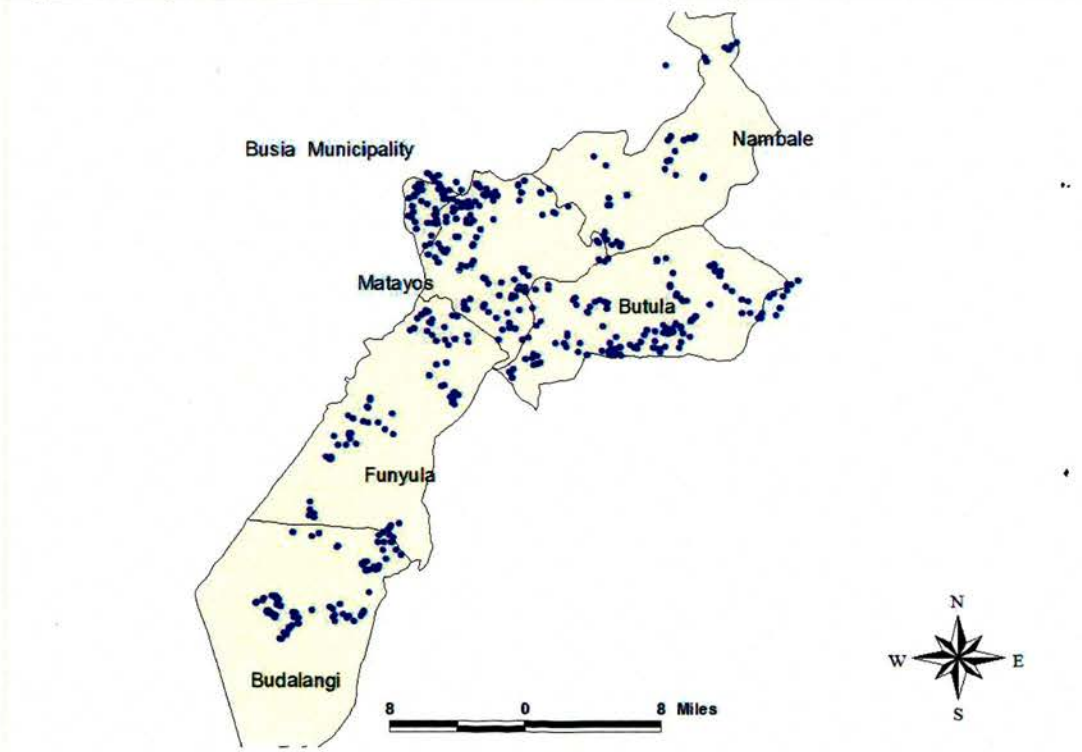


Figure 7: Map of Busia District with location of study subjects



2.2.2 Human Demography

According to the latest statistics from the Ministry of Gender, Children and Social Development, Busia District has a population of 271,173 females and 213,065 males. Life expectancy in 2005 for females was 46.6 years for females and 41 years for males. The majority of the population is Luhya speaking (of which there are many different sub-groups – Idaho, Samia, Ikhosa etc) and is of Bantu ethnicity. The district borders Siaya District, which is made up mainly of Luo speaking lowland Nilotics. There is a reasonable amount of intermarriage between the two groups. The % of households that are female-headed is 30.6%. There are 102,370 women of child bearing age (15-49yrs) (Government of Kenya, 2008). The urban population was 154,348 in 2002 and was expected to rise to 184,678 by the end of 2008. The majority of the urban population lives in Busia Town (57,863) (Government of Kenya, 2002).

Table 7: Population Densities by Division - Busia District

Division	1999				2002			2008	
	Area (km^2)	Population	Density		Population	Density		Population	Density
Budalang'i	306.5	53 356	174		58 363	190		69 836	228
Butula	245.2	95 489	389		104 450	426		124 970	510
Funyula	281.2	73 875	263		80 808	287		96 687	344
Matayos	173.7	55 186	318		60 365	348		72 227	416
Nambale	232.5	67 544	291		73 883	318		88 401	380
Township	22.2	25 158	1 133		27 519	1 240		32 926	1 483
Totals	**1 261.3**	**370 608**	**294**		**405 389**	**321**		**485 047**	**385**

Source: (CBS, 1999) and District Statistics Office, Busia, 2001.

There is widespread poverty in Busia District and the majority of the population lives in food poverty and hardcore poverty – 61.4% and 50.6% respectively. According to the Welfare and Monitoring Survey (WMS III) of 1997, the prevalence of overall poverty in the district was 66% of the population. This poverty manifests itself in various forms: malnutrition amongst children, inability to pay for health care, poor

general health of the population, few assets, few or no livestock, land that barely sustains subsistence as it is mineral and nutrient depleted but not replenished, brewing and partaking of illicit brews and loss of faith in future (Government of Kenya, 2002).

2.2.3 Climate

The rains fall in two distinct periods during the year, the long rains starting in March and continuing to May, and the short rains season starting in late August and finishing in October. The mean annual rainfall for the district is 1,500 mm with most parts of the district receiving between 1,270 mm and 1,790 mm. Budalang'i and Funyula Divisions are closest to Lake Victoria and receive annually between 1,020 mm and 1,270 mm. Budalang'i experiences periodic flooding during the rainy seasons and has large dykes constructed to keep the lake from encroaching on the land.

The annual mean maximum temperatures range from 26°C to 30°C while the annual mean minimum temperatures vary between 14°C and 18°C. As the district is very close to Lake Victoria, with some areas of Budalang'i Division only being accessible by the lake, the district records high rates of evaporation – between 1,800 mm and 2,000 mm per year (Government of Kenya, 2002). Humidity levels are therefore high and species such as riverine tsetse flies *Glossina fuscipes fuscipes (G. f. fuscipes)* flourish and Busia District forms part of a continous tsetse fly belt which extends into southeast Uganda (Ford & Katando, 1973). It is also the ideal habitat for the *Anopheles gambiae* complex mosquito which is implicated in the spread of malaria in Africa and prefers areas in which annual rainfall is above 1,000 mm (Coetzeė *et al.*, 2000).

2.2.4 Agriculture

The long and the short rains support two distinct cropping seasons, however, crops are grown throughout the year. Crops that take longer to mature are grown in the long rains. These are crops such as maize, sorghum, sweet potatoes, soya bean, cow

peas, green gram lentils and beans. These same crops are grown in the short rains, but with the addition of those that mature more quickly such as kale, simsim (sesame seeds) and sunflower. Sugarcane is also a major crop in the area as well as napier grass, cassava, avocados, oranges and bananas.

The soils within the district are of low fertility and are rocky and stony well-drained red clay. Parts of Nambale and Butula Divisions are more fertile with well-drained deep brownish sandy soils which hold water better. Around Lake Victoria the soils are poorly drained and are heavy clay which is hard to cultivatre and perennially flooded. There have been several attempts by various governments to start rice growing schemes in Budalang'i, with varying measures of success.

The district has approximately 924,200 ha of agricultural land. The average farm size within Busia District is 2.5 ha for a small scale farm, of which there are 44,000. Agriculture contributes 35.4% (KSh 1,820.10) to the average monthly household income of KSh 5,141.80. The total acreage under food crops is 58,165 ha and the total acreage under cash crops is 4,935 ha (CBS, 2000; Government of Kenya, 2002).

2.2.5 Livestock

In Busia District in particular, in the last 5 years, two formal Livestock Censuses have been carried out, one by the Government of Kenya and one by Farming in Tsetse Controlled Areas (FITCA) which produced differing results. There have also been informal surveys conducted by the District Veterinary Officer which produced yet another count of livestock which yielded different results from either of the two formal censuses.

The first Livestock Census was carried out in 2000 and 2001 by the (FITCA) project in Busia District. The census covered both Busia and Teso Districts and produced the following figures 3 and 4 for livestock numbers and poultry numbers respectively in Busia District.

Figure 8: Population of livestock: cattle, sheep, goats, donkeys, pigs and rabbits

Population of livestock: cattle, sheep, goats, donkeys, pigs and rabbits								
District/ Divison	Cattle zebu	Cattle exotic/ crosses	Total	Sheep	Goats	Donkeys	Pigs	Rabbits
Busia	**73111**	**1707**	**74818**	**28194**	**50141**	**2118**	**21280**	**16814**
Budalangi	9823	17	9840	2704	9483	77	812	1628
Butula	23350	235	23585	9175	12582	184	2853	4577
Funyula	7118	89	7207	2666	8738	57	2481	1861
Matayos	11248	115	11363	4705	6526	178	5618	2352
Nambale	12648	88	12736	4082	4115	1091	4050	2697
Township	8924	1163	10087	4862	8697	531	5466	3699

Source: (Mosi & Nyandega, 2002)

The FITCA livestock census reported a total of 21,280 pigs in Busia District in 2000-2001. An informal survey carried out by Dr Murukefu, the Busia District Veterinary Officer, found a total of 24,277 pigs in 2003 in the district. The most recent Livestock Census which was carried out by the Government of Kenya (GoK) in late 2003 but has yet to be published.

Figure 9: Population of poultry - chickens, turkeys, ducks, geese, pigeons and quails

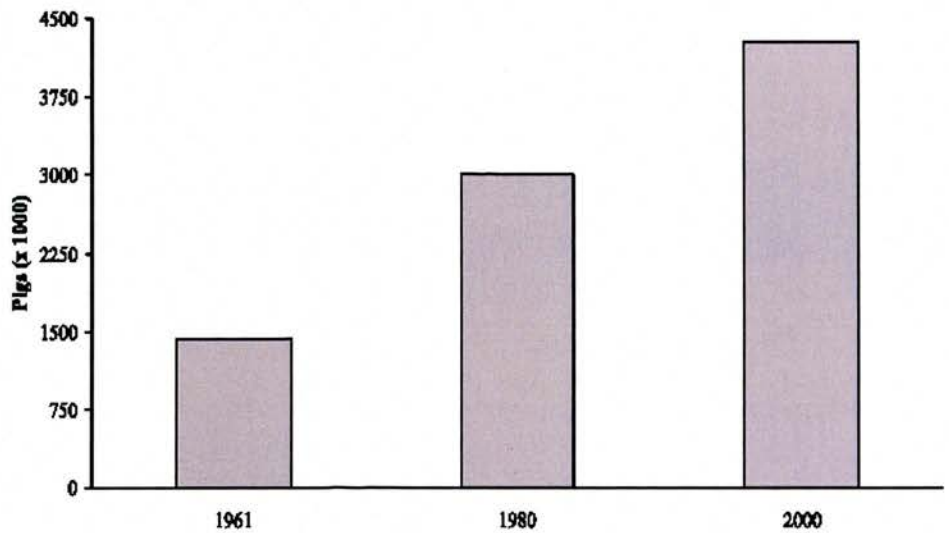
District/ Dvision	Chickens Local	Chickens Exotic	Chickens Total	Turkeys	Ducks	Geese	Pigeons	Quails	TOTAL POULTRY
Busia	592550	17747	610297	7461	46938	2321	13646	7112	**687775**
Budalangi	77491	635	78126	316	11100	112	423	65	**90142**
Butula	165452	826	166278	1594	11675	272	1861	1645	**183325**
Funyula	89340	583	89923	291	3192	240	1797	59	**95502**
Matayos	81213	1439	82652	559	3882	117	790	311	**88311**
Nambale	83800	952	84752	2665	7067	1074	2801	1253	**99612**
Township	95254	13312	108566	2036	10022	506	5974	3779	**130883**

Source: (Mosi & Nyandega, 2002)

As in other parts of sub-Saharan Africa, livestock are regarded as an insurance policy to offset unforeseen expenses or as a ready source of cash to pay for medical expenses or school fees. Small livestock such as sheep or goats and poultry are readily sold to pay for school fees, uniforms, household items of necessity, however, cattle are only sold as a last resort, when the expense is too large to be met by the former livestock (Thuranira, 2005).

Pig keeping is on the increase in Busia District as well as other areas of Western Kenya. According to livestock surveys conducted 18 months apart during 2001-2002 indicated a 171.9% increase in the number of pigs compared to 13.5%, 8.5%, 3.5% and 11.8% increases in cattle, sheep, goats and poultry, respectively (Thuranira, 2005). The figure below illustrates the increasing trend within Eastern and Southern African countries in the pig populations.

Figure 10: Trend in the total pig population in the Eastern and Southern Africa countries of Uganda, Tanzania, Kenya, Zambia, Zimbabwe and Mozambique over the past 40 years



Source: (FAO, 2002)

In these communities pigs are considered low-input livestock which do not require the same level of care as cattle, for example. In addition, pigs are omnivorous; and accomplished scavengers which can grow to market size on minimal feed inputs from the farmer. Smallholder pig production in western Kenya is one of the livelihood strategies used by the local communities as a way to generate income in this poverty prone area.

2.2.6 Constraints to Livestock Production

The Busia District Development Plan (2002-2008) lists some of the constraints to livestock development as being: limited genetic potential of the zebu herd (zebu are

the traditional cattle of the region which are small and require few veterinary inputs to stay alive, however yield very little in terms of milk and meat), poor animal nutrition due to inadequate roughage, inadequate extension coverage, high incidence of animal disease, poor market organisation and traditional animal husbandry practices limiting potential genetic diversity offered by artificial insemination (Government of Kenya, 2002). In addition, "lack of credit or capital" has been identified by the poor as one of the biggest constraints to acquisition of livestock (LID, 1999). These constraints are described in further detail below.

2.2.6.1 Disease

The poor in the developing world are particularly at risk from livestock disease. This is largely because the areas of the world in which the majority of the poor live are within the tropical and sub-tropical regions where climates and eco-systems play host to a number of parasitic infections, many of which are not found in the more temperate climates of the world (Perry *et al.*, 2002). Poverty also contributes to the livestock themselves being more vulnerable to disease. According to a study which looked at the delivery of veterinary services to the poor in Kenya, "few herders and farmers were spending close to the estimated 'ideal' on livestock drugs and the majority of expenditure was on curative rather than preventative treatments. Although apparently willing, the ability of the poor to pay for treatments appears to be a limiting factor. Knowledge regarding livestock health was poor, further contributing to the overall low uptake of veterinary goods and services. Both access and the quality of advice regarding the use of livestock drugs were considered problematic" (Heffernan & Misturelli, 2003). Many resource-poor farmers cannot afford or do not have access to public and private veterinary interventions or the necessary feed inputs to keep their animals in sanitary, disease-free conditions. Poor animal health has been recognised as the most important constraint to livestock production in Africa (WIIAD, 1992) with disease estimated to cause loss of animal livestock output of up to 30%, which is twice that observed in developed countries (FAO, 1990).

Epidemic diseases of livestock such as rinderpest and foot and mouth diseases are considered diseases of trade and therefore receive a lot of public sector attention and involvement in treatment. Endemic diseases such as helminth diseases, trypanosomiasis and East Coast fever have the greatest effect on small scale farmers as although the losses may not be as dramatic as those of epidemic diseases, they are continually present within livestock and the attrition of this disease makes them significant not only at the community level, but also at the national level if one takes into account the aggregation of loss to disease (Perry *et al.*, 2002). Zoonotic diseases such as *Taenia solium* attributed cysticercosis and *Mycobacterium bovis* tuberculosis cause both productivity losses and human health impacts but their impact is largely felt in the public health sector even though the high level of parasitosis present in pigs can cause long term economic losses such as those found in other endemic diseases and is often not noted as significant.

2.2.6.2 Grazing

In Busia District, animals are usually tethered within compounds or taken individually to communal grazing lands. Unlike Uganda, herds are not grazed communally. As the average farm size is 2.5 ha, the competition for grazing and crop production is intense and grazing is land is often poor (Government of Kenya, 2002). Communal grazing lands are located around land that is not deemed productive, is frequently overgrown and near swamps, rivers or streams. This puts animals at risk for trypanosomiasis and other diseases (Murray *et al.*, 2004) and poor grazing leads to poor nutritional status, reducing milk yields, draught power, weight gain and lowered productivity (Perry *et al.*, 2002).

2.3 Background to Taenia solium *porcine cysticercosis and neurocysticercosis in Busia District*

2.3.1 Perception of Cysticercosis in Busia District

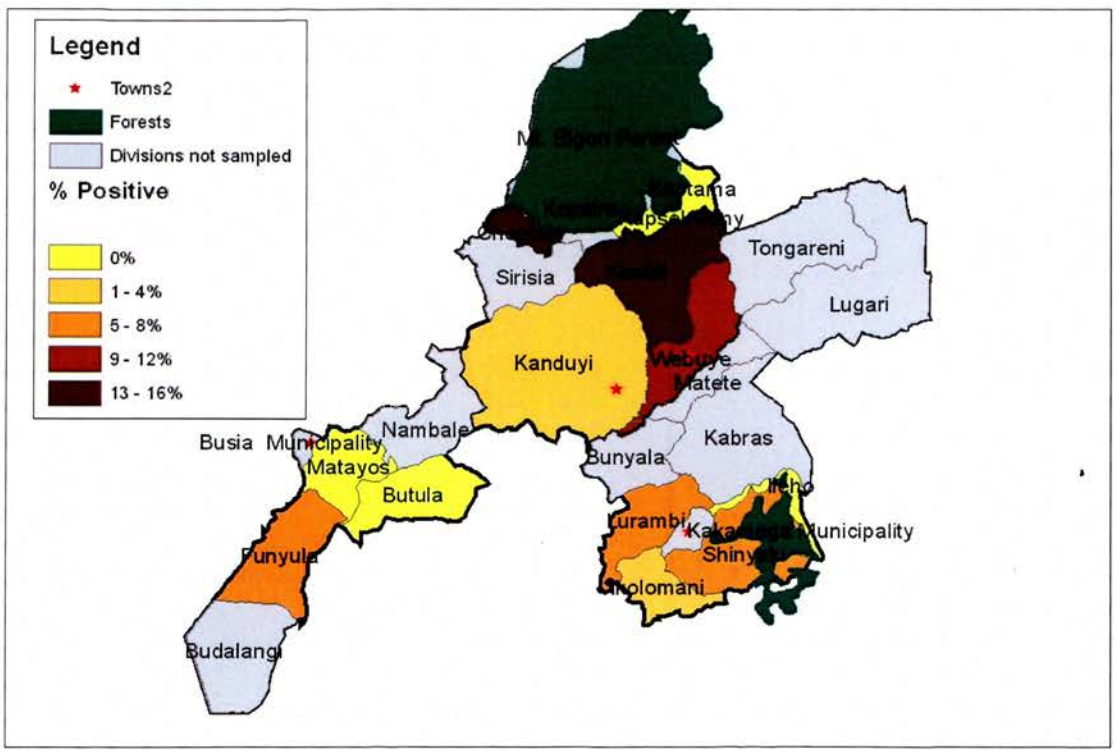
In 2005, DFID funded a study to determine the prevalence of porcine cysticercosis in some districts in Western Kenya, including Busia District. In total, 250 households were surveyed. A few (18%; 46 / 250) of the respondents had heard of a disease in

pigs that could be transmitted to humans, most of whom could not report the exact disease(s) though some made guesses. Among the guesses mentioned were ectoparasites, foot infections, skin infections, and general worm infections. A total of 44 farmers reportedly slaughtered pigs in their compounds, 23 farmers did not have the slaughtered pigs inspected (Mutua *et al.*, 2006).

2.3.2 Cysticercosis in Pigs

In this same study, a total of 320 pigs from 250 households were examined for the presence of *T. solium* larval cysts on the ventral side of the tongue. Out of these, 16 tested positive indicating an estimated prevalence of 5% (16 / 320) using the lingual method of detection. This map indicates the percentages of positive pigs in the individual divisions within the sampled districts.

Figure 11: Proportion of positive cases (prevalence) by division



Source: (Mutua *et al.*, 2006)

In Busia District (divisions Butula, Funyula and Matayos are highlighted in red in the table below), a total of 100 pigs were sampled and of these, 2 were positive for the

presence of cysts in the tongue using lingual examination. All of the positive cases were found in Funyula Division, indicating the percentage of positive pigs for that division to be 6% (2/29). The table below summarizes the number of pigs sampled and the lingual palpation results by division.

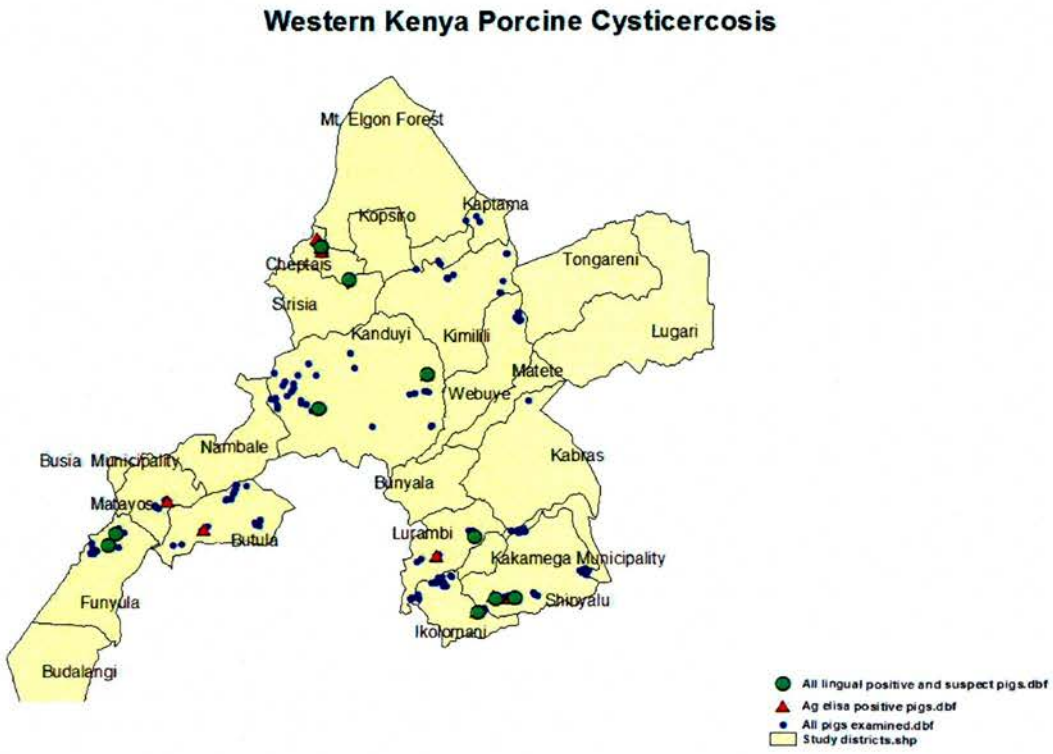
Figure 12: Results of lingual examination for porcine cysticercosis by division in the sampled districts

Division	No of households visited	No of pigs examined	No of pigs positive	% positive
Bumula	25	29	1	3.4
Butula	31	52	0	0
Cheptais	16	19	3	15.7
Funyula	19	29	2	6
Ikolomani	24	26	1	3
Ileho	18	20	0	0
Kanduyi	16	22	0	0
Kapsokwony	2	3	0	0
Kaptama	5	7	0	0
Kimilili	18	25	4	16
Lurambi	15	17	1	5.8
Matayos	15	19	0	0
Ndivisi / webuye	22	22	2	9
Shinyalu	24	30	2	6.6
Total	**250**	**320**	**16**	**5.0**

Source: (Mutua *et al.*, 2006)

In a more recent study, the same subject pigs (320) were tested for porcine cysticercosis using an Ag ELISA. The results of this testing and the lingual examination are found in the map below. The prevalence of porcine cysticercosis by Ag ELISA was (5/320) 1.6%.

Figure 13: Results of Porcine Ag ELISA test



Source: (Mutua *et al.*, 2006)

2.3.3 Perception of Epilepsy in Busia District

Epilepsy is thought to be a disease indicating possession by the devil in Busia District. Epileptics are often kept away from the rest of society and sometimes physically shut away. Epilepsy is known as the "burn disease" as many patients have fallen into open fires and bear scars from burning.

At the Busia District Hospital, treatment for epilepsy falls under the Psychiatric Nurse. It is not thought of as a neurological illness, but rather a psychological illness. If parents are unable to cope with a child's epilepsy, the child is often sent away to school at a Special School which deals with mental disorders and the physically challenged. Children who are on a therapeutic drug regime are often incapable of attending mainstream schooling as teachers claim they fall asleep too often and are not capable of following the same curriculum as the other children.

Chapter 3 - Methodology – Study Design and Diagnostics

3.1 Study Design and Sample Size

3.1.1 Ethical Approval

Ethical approval for this study was obtained from the Kenya Medical Research Institute and is found in Appendix A.

3.1.2 Purpose of study

The study had two major objectives, firstly to estimate the prevalence of seropositivity for neurocysticercosis (NCC) in epileptics in Busia, Kenya and secondly to investigate the risk factors associated with seropositivity to NCC in the same population.

3.1.3 Study design

To estimate seropositivbity for NCC we first identified a population of epileptics and then obtained blood samples for serology. To investigate risk factors, a questionnaire was administered to the study population. On the basis of serology, epileptics were divided into a group with NCC related epilepsy and a group of without NCC related epilepsy, and these were compared with respect to hypothesized risk factors. Risk factors included those which have an impact on the acquisition of porcine cysticercosis and therefore neurocysticercosis and also risk factors are related to poverty. A robust indicator of wealth/poverty was included as an independent risk factor. This latter aspect was achieved through the results of a poverty index.

Null-Hypotheses

1. The prevalence of seropositivity for NCC in a population of epileptics is small or negligible (<5%).
2. There is no significant association between hygiene and sanitation practices (source and treatment of household water, presence and use of latrine, ingestion and preparation of pork), household demographics (age, education level, presence of tapeworm carrier in household), and seropositivity for NCC detected in the population of epileptics.

3. There is no significant association between factors associated with pig keeping (occupation, grazing practices, types of feed, inspection of carcass after slaughter), and knowledge of tapeworm (nodules observed in humans and pigs, observation of passing of proglottids) and seropositivity for NCC detected in the population of epileptics.

3.1.4 Sampling strategy

Our objective was not to estimate the prevalence of epilepsy in Busia, but rather the prevalence of seropositivity for NCC among people with epilepsy. There is no comprehensive list of epileptics in Busia and because epilepsy is a diseases associated with stigma, it is difficult to identify sufferers. Our sampling strategy was to collect information on as many epileptics as was feasible given the time and resources and then to adminsister a questionnaire to, and take serum samples from, all epileptics identified and willing to participate in the study. In all, 1051 epileptics were identified, 423 took part in the questionnaire survey only, 437 took part in the serosurvey only and 191 in both.

3.1.5 Power estimatation

Because of the difficulty in establishing the location of epileptcs and the absence of a sampling frame it was not possible to conduct probabalistic sampling. However, given the sample size (n) of 614, plus an estimate of the total population (N), and an estimate of prevalence (p), and a confidence level (z) of 95%, it is possible to calculate the precsion (e) or margin of error of the estimate.

There was little information on expected prevalence of NCC among epileptics as this was the first serosurvey. While recent reviews have noted the absence of well-conducted studies that can establish the importance of neurocysticercosis in epilepsy (Pal *et al.*, 2000), it is considered the major cause of acquired epilepsy in Latin America (Garcia *et al.*, 1993), and may also be so in sub-Saharan Africa (Dumas, 1989), (Nsengiyumva, 2003). In Nsengiyumva's study carried out in Kiremba, Burundi, of the cases (people with epilepsy), 59.6% were positive for ELISA

cysticercosis serology and of the controls (those without diagnosed epilepsy), 31.5% were positive for cysticercosis. The sample size in this case was 324 cases and 648 controls. In a similar study conducted by Newell in Bururi, Burundi, 4.9% of the 103 epileptic cases and 4.2% of the 72 controls showed exposure to *T. solium* in Ag ELISA (Newell *et al.*, 1997). Where there is doubt about the expected proportion, but it is believed not to be very small (i.e. less than 0.05) it is best to err towards 0.5 as this leads to a larger sample size. Accordingly, the prevalence was assumed to be 0.5.

When the sample is large relative to the total population size (one twentieth or more of the total population) than an adjustment can be made for a finite population. As indicated in Table 3, prevalence rates for epilepsy in sub-Saharan Africa range from 5.2 to 74.4 per 1000 using different sample sizes in door-to-door surveys. The prevalence rate for Kenya of 18.2% (Kaamugisha & Feksi, 1988) is based on one study in Nakuru which is similar to the average prevalence rate for all studies in sub Saharan Africa 1.58%. Given a population of 405,389 people in Busia, a population of 8,828 epileptics is therefore anticipated.

Rearranging and solving the formulae for sample size calculation (n) and sample size adjustment for a finite population (n') it can be concluded that with a sample of 614 epileptics from an estimated population of 8,828 the prevalence of seropositivity for NCC with a precision (or margin of error) of 3.8% can be estimated.

$$n = \frac{Z^2 p(1 - p)}{e^2}$$

$$n' = \frac{n}{1 + \frac{n}{N}}$$

This assumes that there was no systematic bias which would make the sub population of 1051 epileptics identified for this study different from the population of epileptics not identified.

3.1.6 Selection of Epilepsy Patients

Records were obtained from five centres within Busia District to make up the numbers of 500 patients. The records were obtained from St. Martin's School for the Physically and Mentally Disabled in Kisoko, Nangina Special School for the Mentally Handicapped, St Catherine's Special School, Port Victoria Sub-District Hospital and Busia District Hospital. Clinical Officers were also consulted at various government health centres to ensure those on therapeutic drug regimes for epilepsy but not attending the Centres or Schools previously mentioned were not missed. The patients were selected on the basis that they were on a standard regime of epilepsy drugs: phenobarbital, phentoin, and carbamazapine (in some cases, all drugs were being taken, in others one or two of the cocktail). Information such as name, age, sex and village of origin was obtained.

3.1.7 Administration of the Questionnaire (CWGESA) and household wealth

At the next stage of the investigation, those patients previously traced were found again and a questionnaire that has been standardized by the Cysticercosis Working Group for Eastern and Southern Africa (CWGESA) – Appendix B – was given to the epileptic subjects to assess risk factors for neurocysticercosis or taeniasis, which included household demographics, pig husbandry practices, presence of other epileptics, subject knowledge of *Taenia solium*, tapeworms, hygiene and sanitation practices, education levels, occupation and other factors. Not all of the same patients who were identified in the first selection round could be found again but the questionnaire was administered to 500 people plus an additional 128 for a total of 628 subjects.

A second questionnaire to determine indicators of household wealth was also given (n=625). This consisted of 19 aspects of household wellbing including: clothing,

food, health, housing, furniture, utilities, transportation, possessions, domestic employees and income. Each was divided into three categories of Very Poor, Not So Poor and Non Poor based on objectively verifiable, locally relevant indicators. For example, under the housing indicator:

Very poor: Thatch roof, no door, cloth to cover entrance

Not So Poor: Part of roof is mabati, solid front door, one floor is cement

Non Poor: All of roof is mabati, two doors, all house is cement floor

Figure 14 contrasts some characteristics of each group.

Figure 14: Poverty Assessment Checklist Example

Very Poor	Not So Poor	Non Poor
Sleeps on a mat on the floor	Sleeps on a mattress on the floor	Sleeps on a mattress on a bed
Has no shoes	Wears repaired shoes	Wears good shoes
One meal a day	Two meals a day	Three meals a day
No children in school	Boy children in school	All children in school
No radio	Old or damaged radio	New radio
No domestic servants	No domestic servants	Domestic servants
No income or day labour	Part time employment	Full time employment

3.1.8 Collection of serological samples

Using the same subjects previously identified from the questionnaire administration, efforts were made to trace these subjects using the GPS coordinates provided previously. Unfortunately, as the District has no road maps, it was impossible to align homesteads accurately with GPS coordinates and reach them so that not all of the subjects to whom the questionnaire had previously been given were able to be re-traced. For this reason, more subjects were selected using the Key Informant methodology – people of stature within the community were asked to identify epileptics – and the snowballing technique, which is a technique for developing a

research sample where existing study subjects recruit future subjects from among their acquaintances (Sikasunge *et al.*, 2007). Using these techniques, a total of 614 had serum collected from them by medical personnel from the Busia District Hospital.

3.1.9 Subjects with Serological Test and Questionnaire

Owing to the inability to trace all of the previous patients to whom the questionnaire was initially administered, the sample size to whom both the questionnaire was administered and serum collected was 191 subjects. Serological diagnostic tests were performed on all 614 samples and risk factor analysis on 628 subjects using the questionnaire, but statistical analysis involving the association of a positive or negative serological test and risk factor information obtained from the questionnaire was only able to be performed on 191 subjects.

3.2 Serological Diagnostic Techniques Performed in this Study

The use of serology to diagnose NCC in humans is subject to debate (Dorny *et al.*, 2003). In most studies, the correlation between positive serology and neurological symptoms and/or lesions indicative for NCC by imaging techniques is poor (Ramos-Kuri *et al.*, 1992). This may be explained by the unpredictable clinical outcome of the infection and the variable immunological response of the human host to infection. Clinical manifestations of NCC are diverse because of the number of lesions, their size and where they manifest and the reaction of the host's immune system (Garcia & Del Brutto, 2000a). As serological tests and imaging techniques such as CT scans and MRI measure different aspects of the disease, in some patients both tests may present different results. For example, patients who manifest with cysts either subcutaneously or intramuscularly may show a positive result when tested serologically, but will not show any cysts in the brain (Erhart *et al.*, 2002). In contrast, individuals with only inactive lesions or those with a single cerebral lesion causing clinical symptoms may test seronegative (Ohsaki *et al.*, 1999), (Wilson *et al.*, 1991).

The general opinion is that consistent diagnostic criteria of NCC should be based on combined neuroimaging studies, serological tests, clinical presentation and exposure history (Dorny *et al.*, 2003). Several studies have shown that high seropositivity rates for cysticercosis are significantly associated with tapeworm carrier clusters and that seropositive persons are significantly clustered within households, particularly, in households in which a member reported a history of having passed tapeworm proglottids, as well as with individuals with a clinical history of seizures (Diaz-Camacho *et al.*, 1990), (Sarti *et al.*, 1992).

Immunodiagnosis is useful in identifying cysticercosis cases in tapeworm carriers and in family members of tapeworm carriers and patients with NCC. Clinical symptoms of NCC generally occur as a result of an inflammatory reaction around cysticerci, and are in this stage usually associated with degeneration of the cysticerci (Garcia & Del Brutto, 2000a). Early diagnosis of NCC by serology is helpful in identifying patients who can be helped with treatment (Dorny *et al.*, 2003)

3.2.1 Serum Sample Collection

A 10 ml blood sample was obtained from each subject using non-anticoagulated Vacutainer® tubes. The samples were immediately put into cold boxes with ice packs. Each sample was centrifuged in the evening at the Busia District Hospital by qualified laboratory technicians and the sera were transferred into cryotubes (Nunc®) and immediately frozen at -20°C until use. The samples were then shipped to the Institute of Tropical Medicine in Antwerp, Belgium to have serological tests performed on the contents.

3.2.2 ETIB for Taeniasis

The EITB assay was performed on 614 serum samples collected from epileptic patients in Busia District as described by (Wilkins *et al.*, 1999) and in Chapter 1 – Introduction. The samples were collected in January 2007 by medical personnel from the Busia District Hospital. The assay uses excretory/ secretory (TSES) antigens

collected from *in vitro* cultured *T. solium* tapeworms. To identify taeniasis-specific antigens, an immunoblot assay with serum samples from *T. solium* tapeworm carriers and cysticercosis patients was employed. Antigens were identified that reacted with antibodies present in serum samples from taeniasis cases and not with those from cysticercosis patients.

3.2.3 Antibody ELISA

This Ab ELISA was performed on the same 614 serum samples collected from epileptic patients in Busia District as was performed the EITB and the Ag ELISA. The screening for Ab was done by the Ab-ELISA as described by (Guerra *et al.*, 1982). The Ag used for coating the polystyrene ELISA plates was a crude soluble extract of *T. solium* cysticerci. The optical density (OD) was measured at 405 nm with a LP 400 spectrophotometer (Diagnostic Pasteur). The reaction threshold was 0.400. The OD values ‡0.400 were considered to be positive. This method has a sensitivity of 86% and a specificity of 92% (Houinato *et al.*, 1998).

The antigen protein concentration was 2.5 µg/mL in 0.05 M carbonate-bicarbonate buffer at pH 9.6, in a volume of 100 UL per well. After overnight incubation at 4"C, plates were washed with 1% (v/v) Tween-20TM in phosphate buffered saline (Sigma) at pH 7.2 (PBST). The positive and negative control sera, and the study sera, were diluted 1:200 in PBST containing 5% (w/v) bovine albumin fraction V (Sigma) (PBSTA) and placed in the plate wells in a volume of 100 PL per well. After incubation for 1 hour at 37°C followed by 3 washings with PBST, alkaline phosphatase-labelled antihuman immunoglobulin (1g)G (Behring) was added in a volume of 100 FL per well. After another incubation for 1 hour at 37°C and 3 washings with PBST, 100 PL of p-nitrophenyl phosphate substrate was added to each well. The enzymatic reaction was stopped after 45 min by addition of 50 PL duplicate dine-hydrogen peroxide substrate solution.

Absorption (OD) was measured at 405 nm with an LP 400 spectrophotometer (Diagnostic Pasteur). To enhance the measurement specificity, OD values at 690 nm

were subtracted from those at 405 nm (AOD) and the difference between the OD values for the test sera and a pool of sera negative for cysticercosis, ($\Delta OD-\Delta OD_{neg}$), was calculated. The reaction threshold was the mean ($\Delta OD-\Delta OD_{neg}$) value of 100 sera from persons with other parasitic and bacterial infections (including syphilis, malaria, toxoplasmosis, distomatosis, hydatidosis, schistosomiasis, ascariasis and lilariasis) plus 3 SD, which was 0.400. Values 20.400 were considered to be positive.

3.2.4 Antigen ELISA

The test was performed on serum collected from epileptic patients in Busia District, at the Institute of Tropical Medicine (Antwerp) under the supervision of Pierre Dorny. The Ag-ELISA, which was initially developed for *T. saginata* cysticercosis (Brandt *et al.*, 1992), was performed as described by (Dorny *et al.*, 2000).

Briefly, pre-treatment of the samples and ELISA were performed as follows: pre-treatment of the serum samples was done by mixing an equal volume of serum and freshly prepared 5% TCA (Sigma, Chemical Co.) w/v in distilled water and incubation of this mixture for 20 min at room temperature. Incubation was followed by centrifugation for 5 min at 10,000*g*. Next, the pH of the supernatant was raised by adding an equal volume of a sodium carbonate/bicarbonate buffer (0.610M) at pH 10.0 to the supernatant. One hundred microliter of this mixture (final serum dilution 1:4) was used in the ELISA. The assay involved coating of polystyrene ELISA plates (Nunc® Maxisorp) with 100 ml of MoAb B158C11A10 (at a protein concentration of 5 mgml−1) in carbonate buffer, pH 9.6 per well; incubation was 1 h at 37°C and overnight at 4°C. After washing with 0.05% Tween 20 in phosphate buffered saline (pH 7.2) (referred to as PBS-T20) blocking was done with 250 ml per well 1% heat inactivated new born calf serum (NBCS, Life Technologies) in PBS-T20 (1 h, 37°C). Then, trichloroacetic acid pre-treated serum samples were incubated, after which a second biotinylated monoclonal Ab (B60H8A4) was added. A conjugate solution (extravidin-horseradish peroxidase) and a chromogen/substrate solution (OPD (o-Phenylenediamine) in citrate buffer and H2O2) followed. All steps, except the application of the substrate, were done in a shaking incubator for 30 min (coating) or 15 min (other steps) at 37°C. The substrate step was incubated in the dark for 15 min.

Plates were washed in between the steps with 0.05% PBS-Tw 20. The plates were read after stopping the reaction with H2SO4, using a spectrophotometer at 492 nm with a reference of 620 nm. The OD of each serum sample was compared with the mean of negative reference serum samples (n ¼ 8) at a probability level of $P < 0.001$ to determine the result using a modified Student's t test (Sokal & Rohlf, 1981). The ELISA values were expressed as a ratio by dividing the OD of the test sample by the OD of the cut-off value. An ELISA ratio >1 was considered positive (Garcia *et al.*, 1998; Dorny *et al.*, 2000).

3.3 Statistical analysis

3.3.1 Estimating apparent prevalence with exact binomial confidence intervals

The apparent or test prevalence according for each individual test was first calculated using a Stata® routine routines for calculating binomial confidence intervals for p that doesn't depend on the normal approximation. This use of exact confidence intervals avoids the problems of having prevalence estimates greater than 0 or less than 1 and results in intervals that are narrower than when the normal approximation is used

3.3.2 Estimating true prevalence by adjusting apparent prevalence for test performance

Next the true prevalence from test prevalence was estimated using sensitivity and specificity estimates from the literature and the following formula:

true prevalence = (AP+sp-1)/(se+sp-1)

Because there were different estimates for sensitivity and specificity in the literature prevalence was calculated under each set of assumptions.

True prevalence and 95% Bayesian confidence intervals (credible intervals) were next calculated for each test using a Bayesian approach (Vose, 2000).

It was conservatively assumed that the number of true positives (s) in the group lay between 0 and 614 and took estimates of sensitivity (Se) and specificity (Sp) from the literature. The likelihood function for the number of true positives (the true prevalence) is a combination of two binomial distributions, as shown in the following equation:

BINOMDIST (s, n, p(Se) + (1-p)(1-Sp), 0)

Where s was the number tested (614), n is the number found positive, prevalence (p) can taken any value between 0 and 100% and Sp and Se are estimated from the literature. This was set up as a spreadsheet model in Excel© and run for 5000 iterations using Latin Hypercube Sampling Monte Carlo simulation (@Risk).

3.3.3 Combining the results of different tests to produce a single estimate of prevalence

The results of the tests were next combined by using different rules. Tests can be interpreted in parallel in two ways. The first, called "the OR rule," yields a positive diagnosis if any testis positive and a negative diagnosis if all tests are negative. The second rule, called "the AND rule," yields a positive diagnosis only if all tests are positive and a negative diagnosis if any test is negative. Under the OR rule, the sensitivity of the combined result is higher than that of either test alone, but the combined specificity is lower than that of either test. With the AND rule, this is reversed: The specificity of the combined result is higher than either test alone, but the combined sensitivity is lower than that of either test.

Finally, a Bayesian approach was used to combine the results of the different tests in order to get a better estimate of prevalence. The approach of Branscum et al., (2005) was followed. It was considered that the two antibody-based tests (Ab and EITB) were dependent because they measure the same biological phenomenon, and the antigen test (Ag) was conditionally independent.

The model was run in WinBugs using the following code:

```
y[1:K, 1:K, 1:K] ~ dmulti(p[1:K, 1:K, 1:K], n)
p[1,1,1] <- pi*SeAg*(SeAb*SeEITB+covDp) + (1-pi)*(1-SpAg)*((1-SpAb)*(1-SpEITB)+covDn)
p[1,2,1] <- pi*SeAg*(SeAb*(1-SeEITB)-covDp) + (1-pi)*(1-SpAG)*((1-SpAb)*SpEITB-covDn)
p[1,1,2] <- pi*(1-SeAg)*(SeAb*SeEITB+covDp) + (1-pi)*SpAg*((1-SpAb)*(1-SpEITB)+covDn)
p[1,2,2] <- pi*(1-SeAg)*(SeAb*(1-SeEITB)-covDp) + (1-pi)*SpAg*((1-SpAb)*SpEITB-covDn)
p[2,1,1] <- pi*SeAg*((1-SeAb)*SeEITB-covDp) + (1-pi)*(1-SpAg)*(SpAb*(1-SpEITB)-covDn)
```

Expert opinion was used to select most feasible values for sensitivity and specificity .

3.3.4 Relation between poverty and seropositivity

Five different poverty indices were constructed reflecting diffeerent intenisties of poverty. The first index was a sum of all the 'Very Poor' ratings across the 21 well being indicators: this is a measure of intense poverty; the second was a sum of all the 'Not So Poor' ratings across the 21 well being indicators, this is a measure of less intense poverty; the third was a sum of all the 'Not Poor' ratings: this is a measure of wealth. Two poverty indices based on the correlation we found between categories were constructed; the development of these is described under the Results Chapter. T-tests were used to investigate the relationship between poverty and seropositivity.

3.3.5 Risk factor analysis

All data on households and putative risk factors were analysed for their association with prevalence of taeniasis and cysticercosis. First a causal diagram was constructed to help see how risk factors might relate to the dependent (outcome) variable of seropositivity. Next, univariate analysis was conducted to examine the effect of each variable separately by calculating odds ratios (OR), their 95% confidence intervals (95% CI) and *P*-values using maximum likelihood methods. Possible confounding factors were next investigated by constructing two way tables and checking for statistical association using chi square tests.

Afterwards, multivariate analysis (logistic regression) was carried out. Data were first checked for clustering by geographical area using one way anova to calculate the intra-cluster correlation coefficient. This is a measure of lack of indedependence of putatively clustered data. The model was built using those variables that were both causally linked to the outcome variable (seropositivity) and were significant at $P < 0.1$. Factors were added one at a time and those which resulted in improved model

performance retained. Biologically plausible interactions between dependent variables were also checked for. Model diagnostics were carried out to check for any violations of the assumptions underlying logistic regression.

Missing data for the dependent data was dealt with as follows: The missing data was first evaluated in order to assess if it was missing at random, missing completely at random or missing not at random. This was done by summarising all variables according to whether test results were present or absent and comparing these chi^2 for dichotomous variables and t-tests for numerical variables tests to see if the difference was significant. It was assumed that if there were no important differences in between those epileptics with test results and those without test results then the test results could be considered missing at random.

3.3.6 Multiple Imputation by Chained Equations (MICE)

In general, multiple imputation techniques require that missing observations are missing at random (MAR) or missing completely at random. The idea of multiple imputation is to create multiple imputed data sets for a data set with missing values. The analysis of a statistical model is then done on each of the multiple data sets. The multiple analyses are then combined to yield a set of results. Chained equations method uses a series of conditional distributions, e.g. if we have a continuous X, a count variable Y, and a binary Z, and some data for each are missing, we set up (1) a linear regression of X on Y and Z, (2) a Poisson regression of Y on X and Z, and (3) a logistic regression of Z on X and Y. We start by fitting (1) to the observed data, then simulate any missing X from that model. Then we fit (2) using observed Z and X (with missing values filled out by the simulations), and simulate any missing Y. Then we fit (3) using X and Y (with missing values filled by the simulations). We go through multiple iterations, fitting each model in turn, and updating the simulations with each iteration, waiting for the model to converge. We do that multiple times producing multiple datasets.

The drawback is that the conditionals may not specify a unique joint distribution which can make the inferences problematic; but the simulation studies suggest it often works quite well, so it is increasingly used.

All statistical calculations were performed with Stata (Version 10, Copyright 1996–2009 StataCorp LP).

Chapter 4: Risk Factor Analysis, Serology Results and Prevalences

4.1 *Introduction*

The chapter describes the data collected, then presents the results on prevalence of seropositivity for neurocysticercosis (NCC) in epileptics in Busia, Kenya and the risk factors associated with seropositivity to NCC in the same population.

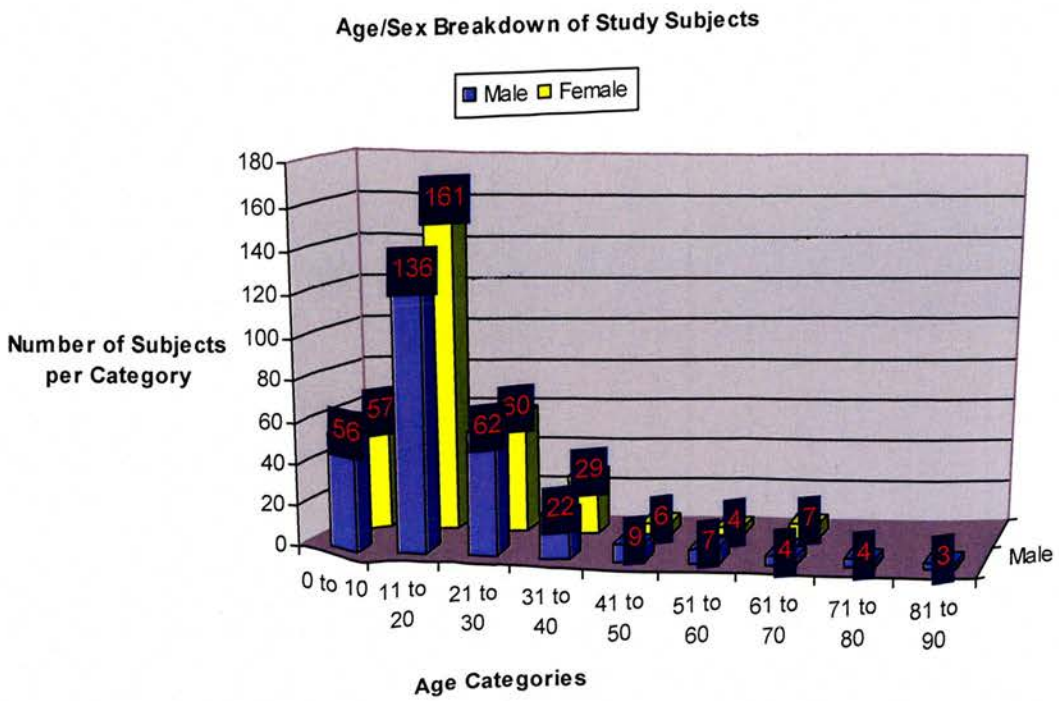
4.2 *Descriptive analysis*

In total, 628 respondents answered the detailed questionnaire on risk factors and other household and individual information. The section that follows gives a characteristic profile of an epileptic living in Busia District.

4.2.1 Age/Sex Breakdown

The following figure represents the age/sex breakdown of the study subjects. The most populous category is 11-20 year olds for both sexes with 21-30 being next. During the study period of 2 years, 6 patients died, some of them from causes related to their having epilepsy such as falling down in a river and drowning or falling and hitting their head on a rock, resulting in death eventually.

Figure 15: Age/Sex Breakdown of Study Subjects



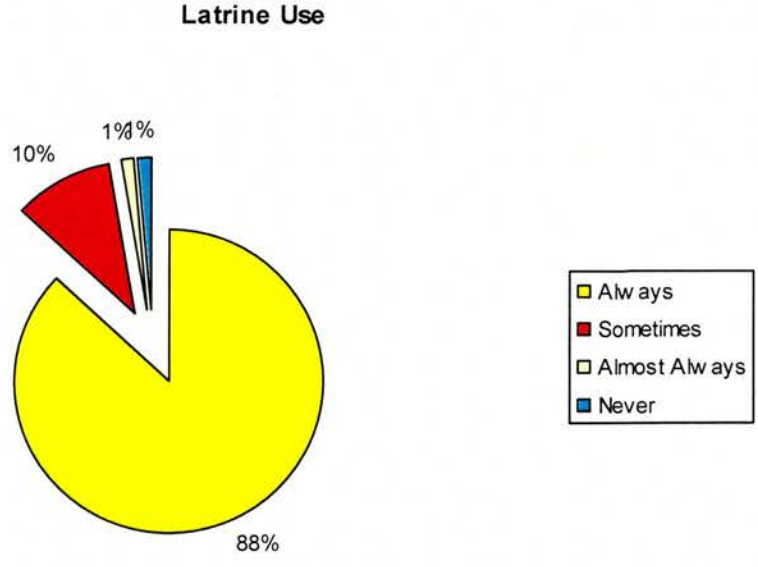
4.2.2 Education

Most epileptics surveyed had completed Primary education (304/592) 51%. Of those who responded to the questionnaire, almost 19% had completed secondary education or higher and 32% had no education.

4.2.3 Hygiene and Sanitation

Of the 605 patients interviewed, the majority (499/605) responded that they had a pit latrine. 106 did not. Of the 499 respondents who had a pit latrine, 88% used it always, 1% used it almost all of the time, 10% used it some of the time and the remaining 1% never used it.

Figure 16: Latrine Use by Respondents



Respondents were asked about the source of their drinking water. Out of 605 asked, 603 responded, the majority of whom used a borehole (44%) Some of the respondents (75/602) 12% live relatively near to urban areas (Busia Town primarily) so are able to have piped water from the Busia Town Council. A further 12% obtain their water from a well and the remaining respondents (31%) get water from the river. Of those whose source of water was the river, 78% of respondents did not treat their water by boiling or other means. 88% of those who obtained water from a well also did not boil or treat their water. 27% of those who obtained water from a borehole boiled water always, almost always or sometimes and 31% of those who

obtained drinking water from the tap boiled water. It can be hypothesized that not boiling water could be an indication of poverty as anybody who has access to tap water lives in an urban centre and is therefore not as poor as those living in rural areas who do not have access to piped water.

4.2.4 Pork Consumption

Of the 89% (464/520) of respondents who consumed pork, 69% consumed pork at least once a month, and 13% ate pork at least once a year. The preferred method of cooking was frying (342/441) 78% while the same (22%) percentage of respondents equally also boiled or barbecued their pork.

4.2.5 Pig Husbandry Practices

Of the 602 respondents, 26% or 157 people kept pigs at the time of the survey. 19% stated they had previously kept pigs at some time in the last five years, while 45% stated they had kept pigs at some point in the past. 97% of all of these pigs ever kept were native pigs. In the planting, growing, and harvesting seasons, the majority (<56%) of farmers tether their pigs. During the fallow season this percentage drops to 46%. For most of the year, slightly over 25% of farmers free range their pigs with this number increasing to 37% in the fallow season. For most of the year, slightly less than 25% of farmers keep their pigs in pens. This number reduces slightly to approximately 17% and 16% of farmers keeping pigs in pens during the fallow and harvest seasons respectively. 60% of the farmers stated they fed their pigs on pasture (foraging), while 97% stated they ate kitchen leftovers. 6% of farmers fed their pigs commercial feed. 15% of farmers kept pigs for home consumption, while 40% kept pigs for reproductive purposes and 61% stated they kept pigs for the purposes of trade. 61% of respondents stated they had never had their pork inspected before or after slaughter, while only 13% stated they always had their meat inspected. 17% stated they sometimes had their meat inspected.

4.2.6 Porcine Cysticercosis

18% of respondents claimed to have ever been told that their pigs or piglets were infected with cysts, and of those, 50% were told in the past year. 31% (178/583) had seen nodules in pig carcasses. These nodules are commonly referred to as "rice" as that is what they resemble when in pig meat. 40% of respondents said the most

common place to find nodules was under the skin of pigs. 66% of respondents did not know how pigs get these nodules.

4.2.7 Human Cysticercosis

78% (469/600) of respondents had heard of tapeworm infection in humans. 41% had learnt about tapeworm infections from a friend or a family member. 53% of respondents said the way one knows if one has a tapeworm infection is by seeing the tapeworm in their faeces, while 4% said fever was a symptom and 13% cited diarrhoea. Respondents were then shown a photograph of tapeworm sections or proglottids and asked if they have ever seen these in their faeces. 46% of respondents stated they had seen segments or proglottids in their faeces, and the course of action upon seeing them was almost equally – 36% and 34% - to seek treatment from a primary health care provider and a pharmacy respectively. 32% of respondents said one gets tapeworm from not washing hands, while 22 % stated that one acquires tapeworm from eating undercooked pig meat. Only 6% said that tapeworm is acquired from contact with another infected person. 76% of respondents to the question said they had never seen nodules or lumps under the skin after being shown a photograph of a subject with obvious nodules showing under the skin.

4.2.8 Epilepsy

79% (486/596) of respondents stated they had ever had headaches lasting more than a few days and 81% (486/599) stated they had seizures characterized by a loss of consciousness or episodes of incontinence or foaming at the mouth. When asked whether they considered themselves to be currently experiencing fainting episodes, 86% (492/589) stated they did. 92% of respondents said they had fainted more than once and 92% said they had experienced uncontrollable twitching or jerking movements of one or more limbs more than once in their lifetime. 57% of respondents said they had experienced a brief onset of hearing or smelling things that were not there or feeling strange body sensations and 92% said this had happened more than once in their lifetimes. 59% (342/581) said they had ever been told they had epilepsy or had had an epileptic seizure while 29% (171/581) said they were not epileptic. 78% (463/594) of total respondents, however, stated they currently had seizures. 92% said they had had more than one seizure. 32% (175/554) stated they

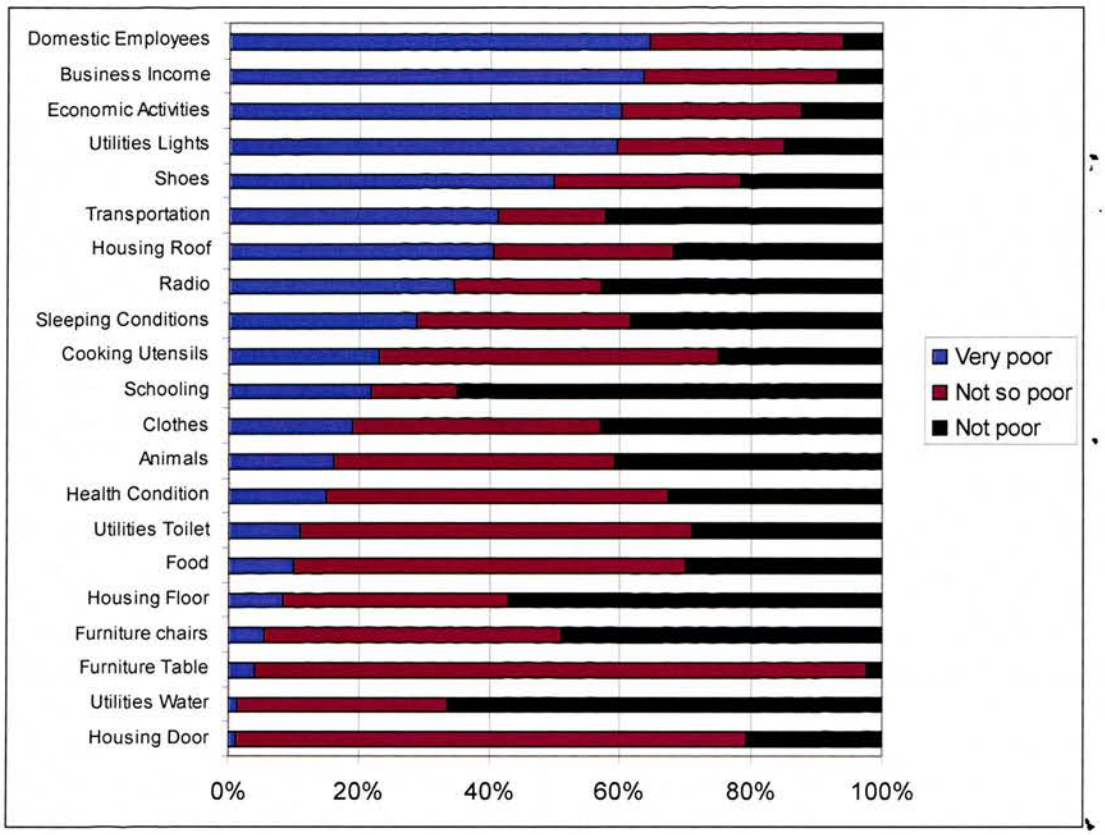
had had a head injury in their lifetime and of those, 99% stated the seizure symptoms they experienced pre-dated the head injury. 16% of respondents claimed to have been ill with meningitis and of those, 63% stated their seizures started after the illness. 21% stated there was another epileptic in the household.

4.3 Poverty levels

The survey of household assets collected information on 21 aspects of household well being (e.g. clothing, housing, transport). The enumerators were asked to observe the items in the checklist (Appendix C) and record their observations. For each of the aspects there were three categories: Very Poor, Not So Poor and Non Poor.

On average, households scored Very Poor on 4.96 indicators, Not So Poor on 6.77 indcators and Non Poor on 5.76 indicators. Figure 17 shows the results for different aspects of household well-being.

Figure 17: Table showing different categories of poverty in study population, based on percentage of those having assets or characteristics according to Poverty Checklist



4.3.1 Poverty index

There is a positive correlation between "very poor" and "not so poor" and a negative correlation between "very poor" and "non poor" (Figure 18). This suggests that 'very poor' and 'not so poor' are both measures of poverty, whereas 'non poor' is a measure of wealth. We constructed two poverty indices: in the first (Poverty Index 1), Very poor was given a weight of 2, Not So Poor of 1 and these were summed to give a measure of poverty.

Figure 18: Correlation between the different well-being/poverty indices

	verypoor	Not So Poor	nonpoor
verypoor	1.0000		
notsopoor	0.3273	1.0000	
nonpoor	-0.1686	0.0524	1.0000

A second poverty index was constructed which gave a greater weighting to the category of Very Poor and a negative weighting to Non Poor. The second poverty index was constructed as follows:

```
Verypoor x 3 + notsopoor x 1 - nonpoor = poverty index2
```

The following table (Figure 19) illustrates the dichotomous representation of the selection of 30% of the population as "Very Poor" (based on a weighting of 3 in poverty index 2), which results in a cut-off value of "8". The density of the population in the categories "Very Poor", "Poor" and "Not so Poor" is shown in Figure 20, following, with the Poverty Index 2 graph showing a "normal" distribution.

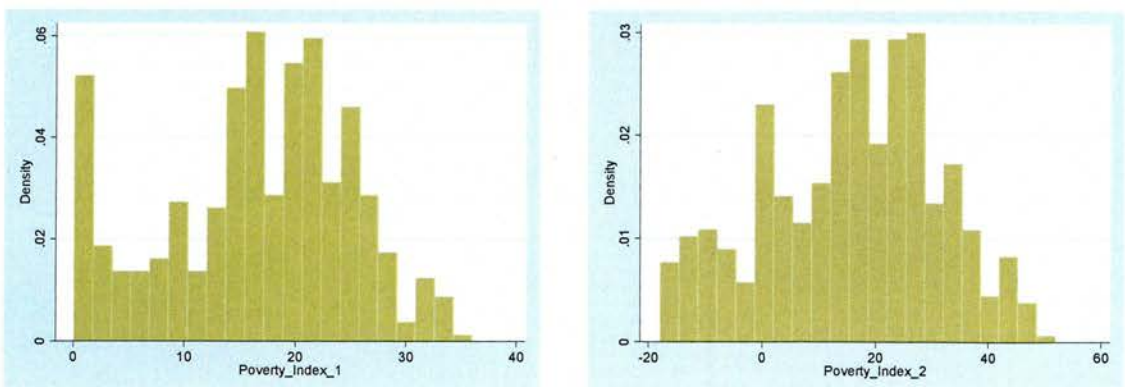
Figure 19: Poverty Index 2 Dichotomous values table showing cut-off value of 8 based on 30% of population calculation

Poverty Index 2	Frequency	Percent	Cumulative Percent
-18	3	0.64	0.64
-17	3	0.64	1.28
-16	2	0.43	1.7
-15	4	0.85	2.55
-14	2	0.43	2.98
-13	5	1.06	4.04
-12	9	1.91	5.96
-11	11	2.34	8.3
-10	3	0.64	8.94
-9	3	0.64	9.57
-8	1	0.21	9.79
-7	5	1.06	10.85
-6	3	0.64	11.49
-5	5	1.06	12.55
-4	2	0.43	12.98
-3	2	0.43	13.4
-2	5	1.06	14.47
-1	8	1.7	16.17
0	23	4.89	21.06
1	5	1.06	22.13
3	12	2.55	24.68
4	6	1.28	25.96
5	4	0.85	26.81
6	6	1.28	28.09
7	5	1.06	29.15
8	7	1.49	30.64
9	6	1.28	31.91
10	12	2.55	34.47
11	6	1.28	35.74
12	10	2.13	37.87

Cut-off point for poverty index2 "Very Poor" at 30% of population

13	9	1.91	39.79
14	13	2.77	42.55
15	9	1.91	44.47
16	10	2.13	46.6
17	20	4.26	50.85
18	16	3.4	54.26
19	9	1.91	56.17
20	14	2.98	59.15
21	7	1.49	60.64
22	12	2.55	63.19
23	13	2.77	65.96
24	11	2.34	68.3
25	10	2.13	70.43
26	15	3.19	73.62
27	13	2.77	76.38
28	19	4.04	80.43
29	6	1.28	81.7
30	9	1.91	83.62
31	6	1.28	84.89
32	8	1.7	86.6
33	7	1.49	88.09
34	5	1.06	89.15
35	7	1.49	90.64
36	4	0.85	91.49
37	3	0.64	92.13
38	10	2.13	94.26
39	2	0.43	94.68
40	3	0.64	95.32
41	2	0.43	95.74
42	2	0.43	96.17
43	3	0.64	96.81
44	5	1.06	97.87
45	3	0.64	98.51
46	3	0.64	99.15
47	2	0.43	99.57
48	1	0.21	99.79
52	1	0.21	100

Figure 20: Density of Study Population in Poverty Index 1 and Poverty Index 2



4.4 Relation between poverty and seropositive test result

For all four measures of poverty, the seropositive group scored higher, and for the measure of wealth (Not Poor) the seropositive group scoed lower; however, none of these relations were significant (ttest).

	Seronegative	**Seropositive**	**p**
Very Poor	4.8	5.2	0.251
Not So Poor	6.6	6.8	0.646
Not Poor	5.7	5.3	0.392
Poverty Index 1	16.1	17.1	0.241
Poverty Index 2	15.2	17.0	0.238

4.6 Sero-prevalence estimates

4.6.1 Estimating apparent prevalence

Serum samples were available from 614 epileptics and three tests were used: two of these were antibody detection tests - enzyme linked immunosorbent assay (ELISA) and enzyme linked imuunoelectrotransfer blot tests (EIBT) - and one antigen test (ELISA). The results of these tests are shown in table 9.

Table 8: Apparent prevalence for *Taenia solium* cysticercosis by three tests

No of samples	*Tests*		
	Ab ELISA	EITB	Ag ELISA
414	-	-	-
184	+	-	-
6	+	+	-
9	-	+	-
1	+	-	+
0	-	-	+
614			

The prevalences and associated confidence intervals for the tests are given in table 10.

Table 9: Prevalences and Associated Confidence Intervals (CI)

	n	*Positive*	*Prevalence*	*Binomial exact 95% CI*

Antibody ELISA	614	191	31.07	27.46	34.94
Antigen ELISA	614	1	1.63	0.004	0.90
EITB	614	15	2.44	1.37	4.00

4.6.2 Estimating true prevalence for each test by adjusting apparent prevalence according to test performance

The tests have varying sensitivity and specificity reported in the literature. When apparent prevalence is adjusted by sensitivity and specificity estimates the following true prevalences are obtained:

Table 10: True Prevalence of Three Tests

	Ab Elisa		*Ag Elisa*		*EITB*
Test positive	191	191	1	1	15
Apparent prevalence	0.3111	0.3111	0.0016	0.0016	0.0244
Sensitivity	0.86	0.93	0.65	0.944	0.98
Specificity	0.92	0.89	0.92	1	1
True prevalence	0.2962 (Prado-Jean et al., 2007)	0.2452 (Sloan et al., 1995)	0.1375 (Garcia et al., 2000)	0.0017 (Erhart et al., 2002)	0.0249 (Tsang et al., 1989)

A maximum likelihood approach to calculating true prevalence by adjusting apparent prevalence for sensitivity and specificity and probabilistically modeling produced the estimates shown in table 12.

Table 11: True Prevalence after Adjusting Apparent Prevalence and Probabilistic Modelling

	Ab Elisa		*Ag Elisa*		*EITB*
Test positive	191	191	1	1	15
Apparent prevalence	0.3111	0.3111	0.0016	0.0016	0.0244
Sensitivity	0.86	0.93	0.65	0.944	0.98
Specificity	0.92	0.89	0.92	1	1
True prevalence	0.2970	0.2460	0.0365	0.0100	0.0264
	0.2575-0.3373 (Prado-Jean et al., 2007)	0.2083-0.2844 (Sloan et al., 1995)	0.0003-0.0085 (Garcia et al., 2000)	0.0032-0.0170 (Erhart et al., 2002)	0.0145-0.0395 (Tsang et al., 1989)

4.6.3 Combining the results of different tests to produce a single estimate of prevalence

Tests can be interpreted in parallel in two ways. The first, called "the OR rule," yields a positive diagnosis if any test is positive and a negative diagnosis if all tests are negative. The second rule, called "the AND rule," yields a positive diagnosis only

if all tests are positive and a negative diagnosis if any test is negative. Using the AND then none of the patients would be considered positive for cysticercosis; using the OR rule then 200 are positive or 32.6%.

Bayesian modelling to estimate the prevalence given three imperfect tests (two conditionally dependent and one independent) in a single population of epileptics gave the results summarised in table 13. The estimate of 0.38% positives is closer to that of the results for the Antigen and EITB antibody tests.

Table 12: Prevalence of *Taenia solium* cysticercosis after Bayesian modelling

Mode	*SeAb*	*SeAg*	*SeEITB*	*SpAb*	*SpAg*	*SpEITB*	*prevalence*
Mean	0.7988	0.7099	0.7845	0.6943	0.9885	0.9761	0.0038
SD	0.1389	0.1634	0.1635	0.0177	0.0041	0.0062	0.0035
MC_error	0.0078	0.0023	0.0098	0.0008	4E-05	0.0002	6.11E-05
val2.5pc	0.4669	0.3509	0.4008	0.6596	0.9792	0.9626	2.246E-4 0
median	0.8285	0.7301	0.8252	0.6942	0.989	0.9764	0.0028
val97.5pc	0.9811	0.9576	0.9875	0.7293	0.995	0.9869	0.0131
start	10000	10000	10000	10000	10000	10000	10000
sample	13001	13001	13001	13001	13001	13001	13001

For this model test sensitivity and specificity were assessed using expert opinion based on the literature reports.

SeAb=0.90, SpAb=0.95, SeEITB=0.95, SpEITB=0.99, SeAg=0.85, SpAg=0.92

4.7 Risk Factor Analysis

4.7.1 Risk factors under investigation

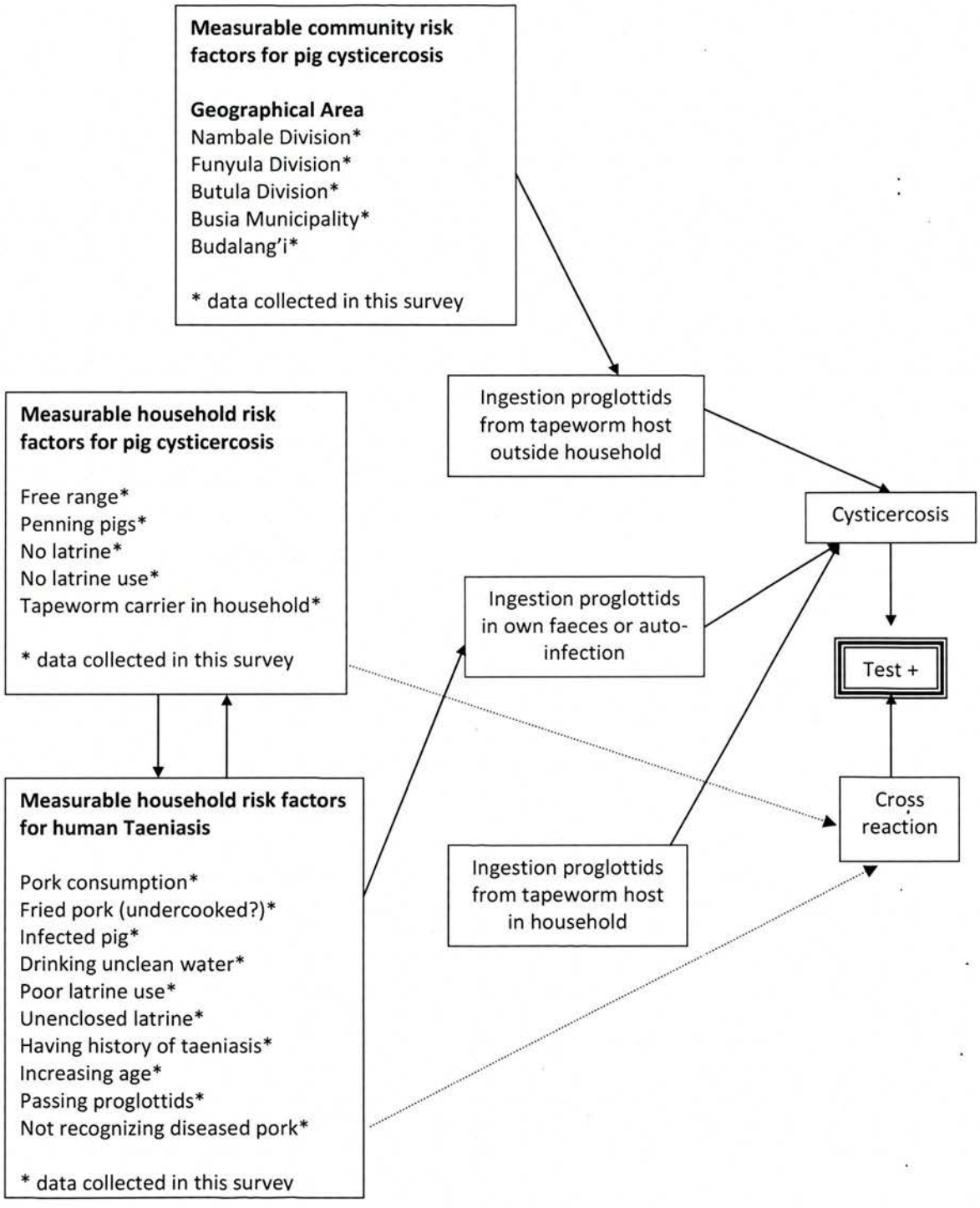
Information was systematically collected for 625 epileptics, on the factors potentially associated with taeniasis and cysticercosis, which are under investigation in this chapter. Data on demographic characteristics, social status and behavioural patterns were collected for cases and controls at the time of the first contact with that individual. Data were recorded on name, age, sex, occupation, education, village of living, eating habits and diet, food preference, drinking unclean water, presence and structure of latrine, the usual site of defecation, pig ownership, general knowledge related to *T. solium* infection and its causation. Respondents were asked whether they had ever heard of or seen cysticerci containing pork and shown a photograph of the same. They were also asked if they had seen nodules under the skin of humans and shown a photo of the same. Questions were asked based on a structured questionnaire approved by the CWGESA (Cysticercosis Working Group of Eastern

and Southern Africa), and data regarding latrine construction and pig-raising practice were obtained by direct observation. Data on household assets was collected by a separate questionnaire.

4.7.2 Causal diagram

A causal diagram was developed using factors which logically should be a risk for both pig cysticercosis and human taeniasis, based on the extensive literature review reported earlier. These factors could be linked through human seropositivity for NCC through directly or indirectly as shown in the diagram. Moreover, seropostivity may be the result of cross-reactions resulting from other helminth or protozoal infections in people and many of the risk factors for these are also risk factors for taeniasis and cysticercosis (e.g. poor latrine use, drinking unclean water, farming as an occupation).

Figure 21: Causal Model for Risk Factors for Taeniasis and Cysticercosis



4.7.3 Univariate Analysis Odds Ratio

For the univariate analysis, the dependent variable was sereopositivity according to any test (the OR rule). All data for which household information was gathered was analysed. There were 222 variables in all, for which 211 there were enough

responses to construct odds ratios.The odds ratios for the 22 risk factors that were found to be significantly associated with increased or decreased risk of seropositivity at P <0.1 are given in table 14.

Table 13: Odds Ratios for Significant Risk Factors in Univariate Analysis

Variable	Cases (n=81) Exposed	%	Controls (n=129) Exposed	%	Odds Ratio	95% CI	*P*
Fever a sign of tapeworm infection	9	11	1	1	15.5	(2.01-686.05)	0.001
Latrine used	39	48	86	67	0.4	(0.18-0.81)	0.006
Seen nodules under the skin	6	7	1	1	10.6	(1.23-490.09)	0.008
Farmer	11	14	6	5	3.7	(1.14-12.85)	0.012
Other epileptic in household	10	12	5	4	3.6	(1.05-13.97)	0.018
Boils drinking water	13	16	8	6	3.0	(1.05-8.54)	0.02
Eats pork	9	11	4	3	3.9	(1.01-17.60)	0.022
Knows one can acquire tapeworm from eating pork	17	21	14	11	2.4	(0.98-5.86)	0.033
Has had a head injury	13	16	37	29	0.5	(0.21-1.01)	0.037
Has had a seizure	12	15	8	6	2.7	(0.93-7.79)	0.039
Does not use a latrine to defecate	20	25	17	13	2.1	(0.95-4.59)	0.045
Never boils drinking water	56	70	103	80	0.5	(0.24-1.10)	0.06
Boils drinking water sometimes	21	26	20	16	2.0	(0.91-4.10)	0.06
Has seen subcutaneous nodules on pigs	33	41	38	30	1.7	(0.92-3.34)	0.063
Feeds pigs commercial feed	3	4	1	1	6.7	(0.48-361.0)	0.072
Feeds pigs feed other than pasture, kitchen scraps or commercial	3	4	1	1	6.7	(0.48-361.0)	0.072
Gets drinking water from the river	20	25	47	37	0.6	(0.29-1.10)	0.074
Has been told has epilepsy	8	10	5	4	2.8	(0.75-11.06)	0.077
Lives in Nambale Division	8	10	5	4	2.7	(0.75-10.92)	0.079
Classification as "Very Poor" in Poverty Index	23	29	24	19	1.8	(0.87-3.62)	0.085
Hallucinates and smells unusual aromas during seizure	4	5	15	12	0.4	(0.09-1.27)	0.088
Never uses the latrine to defecate	0	0	4	3	0	(0.00-1.37)	0.093

For most of the factors the effects on probability of seropositivity were as anticipated. However for some factors, especially those associated with water use the

direction of influence was not as predicted. In particular, never boiling water decreased risk and boiling water sometimes or always increased risk, while drinking from the river decreased risk. Never using a latrine also decreased risk. Eating pork occasionally (monthly to yearly) was more risky than eating frequently (weekly to monthly).

Table 14: Seropositivity and Predicted Risk

	Effect on Risk of seropositivity	*Predicted*
Latrine used (observation)	Decrease	Yes
History of head injury	Decrease	Yes
Never boil water	Decrease	No
Drinks water from river	Decrease	No
Sees or smells things during fits	Decrease	n/a
Never uses a latrine - report	Decrease	No
Consider fever a sign of tapeworm infection	Increase	n/a
Had skin nodules in the past	Increase	Yes
Farmer	Increase	Yes
Other epileptics in household	Increase	Yes
Boil all water	Increase	No
Eat pork monthly to yearly	Increase	No
Know pigs can spread tapeworms	Increase	No
History of seizure	Increase	n/a
Latrine not used (observation)	Increase	Yes
Boil water always or sometimes	Increase	No
Has seen nodules in pigs	Increase	Yes
Feeds pigs commercial food	Increase	n/a
Feeds pigs other than leftovers/commercial/pasture	Increase	Yes
Had epilepsy last year	Increase	n/a
Lives in Nambale	Increase	n/a
Very poor	Increase	Yes

We conducted additional investigation on those factors which seemed to be inconsistent with literature and the causal model.

4.7.3.1 Toilet Use

The questionnaire had 2 sections on use of latrines. People were first asked if they had a latrine at home, and then if they said yes whether they used it always, almost always, sometimes or never. Subsequently enumerators used direct observation to see if latrine was present or not, and if present was it open or closed, and was there evidence of use or no use.

The results are summarised in table 16. There was a strong and significant ($P < 0.1$) decrease in risk when a latrine is observed to be used and an increase in risk (significant at $P < 0.05$) when a latrine is observed to be not used. However, the relation between self-reported latrine use and risk of seropositivity was weak and contradictory. Moreover there was no relation (p=0.205, chi^2 test) between households who claim to use the latrine and observable use of the latrine.

Table 15: Toilet Use as a Significant Risk Factor

Exposure	Total	Exposed	%	Total	Exposed	%	Odds Ratio		p
Observed									
Latrine use (yes)	81	39	48.15	129	86	66.67	0.38	[0.18-0.81]	0.006
Latrine enclosed	81	28	34.57	129	47	36.43	0.86	[0.45-1.62]	0.61
Latrine open	81	9	11.11	129	12	9.30	1.16	[0.41-3.20]	0.745
No latrine present	81	11	13.58	129	16	12.40	1.06	[0.42-2.60]	0.894
Latrine partially enclosed	81	23	28.4	129	35	27.13	1	[0.51-1.97]	0.989
Latrine present but not used	81	20	24.69	129	17	13.18	2.08	[0.95-4.59]	0.045
Respondents Answers									
Latrine use									
All the time	81	62	76.54	129	85	65.89	1.7	[0.57-5.70]	0.298
Almost all the time	81	0	0	129	2	1.55	0	[0.00-2.80]	0.238
Sometimes	81	6	7.41	129	8	6.20	1.1	[0.30-3.82]	0.865
Very rarely	81	6	7.41	129	12	9.30	0.7	[0.20-2.16]	0.5
Never	81	0	0	129	4	3.10	0	[0.00-1.37]	0.093

4.7.3.2 Water use

Boiling water increased the risk of seropositivity in contrast to never boiling which decreased risk. People who never boiled water, however were significantly more likely ($P = 0.008$) to drink water from a well, leading to speculation that this source of water is potentially not contaminated and could be protective vs drinking water from a tap, river or borehole.

Figure 22: Correlation between boiling water and water source

Never Boils	Origin: Well 0	1	Total
0	136	9	145
	93.79	6.21	100.00
	25.86	11.84	24.09
1	390	67	457
	85.34	14.66	100.00
	74.14	88.16	75.91
Total	526	76	602
	87.38	12.62	100.00
	100.00	100.00	100.00

Pearson $chi^2(1) = 7.1318$ Pr = 0.008

There is, however, a degree of ambiguity about the significance of boiling water and the well as an uncontaminated source, as boiling water all the time increased risk and those who did boil all the time were more likely to have well water or tap water as the source. More research needs to be done in order to explain this anomaly.

Figure 23: Association between boiling water all the time and well source

Always boils	Origin: well 0	1	Total
0	453	71	524
	86.45	13.55	100.00
	86.12	93.42	87.04
1	73	5	78
	93.59	6.41	100.00
	13.88	6.58	12.96
Total	526	76	602
	87.38	12.62	100.00
	100.00	100.00	100.00

Pearson $chi^2(1) = 3.1372$ Pr = 0.077

Figure 24: Association between boiling water all the time and tap as source

Always boils	Origin: tap 0	1	Total
0	466	58	524
	88.93	11.07	100.00
	88.59	77.33	87.19
1	60	17	77
	77.92	22.08	100.00
	11.41	22.67	12.81
Total	526	75	601
	87.52	12.48	100.00
	100.00	100.00	100.00

Pearson $chi^2(1) = 7.4501$ Pr = 0.006

4.7.4 Multivariate Analysis

The set of probable correlates identified by univariate logistic regressions and those that were considered to be logically associated with risk were then subjected to multivariate logistic regression. These risk factors included:

Neurocysticercosis

- Eating pork – we included eating pork monthly to yearly as a risk factor as the univarieate analysis suggested this was more predictive than eating mork weekly
- Eating undercooked pork – this was not included because those replying yes also replied yes to eating pork and there was a problem with collinearity
- Other epileptics in the household – this was included as an indicator of common exposure
- Spatial location – this was included as NCC often occurs in spatial clusters
- Farming – this was included as a factor known to increase exposure to human faeces
- Skin nodules noticed more than one year ago – this was included as an indicator they person had consumed proglottids in the past
- Shedding proglottids – this was not included because there were too few responses

Taeniasis

- drinking unclean water – this was not included because univariate analysis suggested the relation between drinking unclean water and seropositivity was unclear
- poor latrine use – this was included as a major risk factor
- raising pigs – this was included
- had seen nodules in pig meat – this was included as an indicator of exposure
- having taeniasis - not included because too many missing variables
- passing proglottids: not included because too many missing variables

The model with retained variables is shown in Figure 25.

Figure 25: Multivariate Logistic Regression Analysis

```
Logistic regression                             Number of obs   =         92
                                                LR chi2(8)      =      27.82
                                                Prob > chi2     =     0.0005
Log likelihood = -44.875906                     Pseudo R2       =     0.2366
```

test	*Odds Ratio*	*Std. Err.*	*P*	*95% Conf.*	*Interval*
No other epileptics in household	0.22	0.13	0.01	0.07	0.68
Lives in Nambale	12.32	14.91	0.04	1.15	131.95
Skin nodules > 1 year ago	13.04	16.79	0.05	1.05	162.55
Farming	4.58	3.62	0.05	0.97	21.58
Eats pork monthly to yearly	5.12	5.21	0.11	0.70	37.67
Latrine not used	3.26	2.50	0.12	0.73	14.67
Kept pigs	1.05	0.58	0.93	0.36	3.08
Has seen nodules in pig meat	1.89	1.05	0.25	0.63	5.60

All factors were consistent with the causal model. People with no other epileptics in the household were at less risk of seropositivity. One of the geographical subdivisions (Nambale) had significantly more seropositives Farming as an occupation and having seen nodules under the skin are significant at $P = 0.05$. The pseudo R squared for the model was 0.24 suggesting it was reasonably predictive.

Diagnostics were then run on the linear regression model: the model was examined for the following assumptions:

- Linearity - the relationships between the predictors and the outcome variable should be linear
- Normality - the errors should be normally distributed - technically normality is necessary only for hypothesis tests to be valid,
  estimation of the coefficients only requires that the errors be identically and independently distributed
- Homogeneity of variance (homoscedasticity) - the error variance should be constant
- Collinearity - predictors that are highly collinear, i.e., linearly related, can cause problems in estimating the regression coefficients
- Independence - the errors associated with one observation are not correlated with the errors of any other observation
- Model specification - the model should be properly specified (including all relevant variables, and excluding irrelevant variables)
- Influence - individual observations that exert undue influence on the coefficients

Diagnostic were satisfactory.

4.7.5 Imputation of missing data

Respondents were divided into those with and without missing test results and then using t-tests and chi-square tests to establish whether the two groups differ significantly.

It was discovered that epileptics who had test results differ significantly from those who did not with respect to:

1. Age: they are significantly older
2. Place: they are significantly more likely to come from Matayos
3. Pork consumption: they are less likely to eat pork less than once a month but more than once a year i.e. less likely to be occasional consumers
4. Pig ownership: more likely to have owned pigs more than 5 years ago
5. Signs of a tapeworm: more likely to say that fever was a sign of tapeworm
6. Observing nodules: more likely to be unable to say when they observed skin nodules
7. They were less likely to say they had not had a fit
8. They were less likely to say they had not had a seizure

Given that 241 variables were compared at a P of 0.05, it would be expected that 12 would be significant by chance alone. Only age, place, and having a fit were highly significant. It is reasonable to assume that people who were older were more likely to complete testing and that people who did not have a fit were less likely to complete testing.

There are no major differences between those who got testing and those who didn't and therefore the missing dependent variables (test results) could be imputed.

Next, using the multiple imputation by chained equations methodology (MICE), the model was run again. The risk factors "Nambale" and "skin nodules seen more than one year ago' had to be discarded as they predict success of the model perfectly. The imputed model drew on 210 observations, whereas the model without imputed data drew on only 92 observations. This model with imputed dependent variables gave similar results to the model which dealt with missing data by case wise deletion, however, this model using imputed data showed known risk factors to be more significant. For example in the previous model, although observing that the latrine was not used had an odds ratio of the 3.26 it was not significant. In the model using imputed data it was significant at P= 0.05.

```
Multiple imputation parameter estimates (5 imputations)
------------------------------------------------------------------------------
        test |      Coef.   Std. Err.      z    P>|z|     [95% Conf. Interval]
-------------+----------------------------------------------------------------
Latrine not
Used
             |    1.119313      .4655     2.40   0.016       .20695    2.031677
No epileptics
in household |   -.5851674   .4304749    -1.36   0.174    -1.428883    .2585479

Has kept pigs|    .1799808   .3659335     0.49   0.623    -.5372357    .8971973

Eats pork at
least 1/year |     1.43617   .8283569     1.73   0.083    -.1873791     3.05972

Is a farmerg |    1.598598   .6443174     2.48   0.013     .3357593    2.861437

Has seen
nodules under
the skin     |    .6555746   .3323315     1.97   0.049     .0042169    1.306932
       _cons |   -.9949981    .366995    -2.71   0.007    -1.714295   -.2757012
------------------------------------------------------------------------------
```

Because the imputed model drew on a larger number of epileptics (210 rather than 92) more independent variables were able to be included in the analysis. However, none of these additional independent variables proved to be significant. The third model is shown below:

Figure 26: Third Model Multivariate Analysis - Multiple Imputations

```
Multiple imputation parameter estimates (5 imputations)
------------------------------------------------------------------------------
        test |      Coef.   Std. Err.      z    P>|z|     [95% Conf. Interval]
-------------+----------------------------------------------------------------
Latrine not
used         |    1.119588   .4564316     2.45   0.014     .2249989    2.014178

Poverty Ind2 |   -.0011918   .0118844    -0.10   0.920    -.0244848    .0221012

Age a factor |    .0048093   .0114069     0.42   0.673    -.0175479    .0271665

Is a farmer  |    1.607055   .6626896     2.43   0.015     .3082069    2.905902

Eats pork at
least 1/year |    1.438533   .8540913     1.68   0.092    -.2354554    3.112521

Kept or keeps
pigs         |     .184198   .3653422     0.50   0.614    -.5318596    .9002555

Has seen tape
worm in faeces    .0612016   .4352565     0.14   0.888    -.7918856    .9142887
No epileptics
in household |   -.6033377   .4215846    -1.43   0.152    -1.429628    .2229529

Has seen
nodules under
the skin     |    .6683051   .3395602     1.97   0.049     .0027793    1.333831
       _cons |   -1.101809   .4804339    -2.29   0.022    -2.043442   -.1601755
------------------------------------------------------------------------------
210 observations (imputation 1).
```

Chapter 5: Discussion

5.1 Objectives

Epilepsy is the most common serious neurological disorder and is one of the world's most prevalent noncommunicable diseases. Over four-fifths of the 50 million people with epilepsy are thought to be in developing countries; much of this condition results from preventable causes (Scott, 2001). The World Health Assembly included cysticercosis, human infection by the larvae of *Taenia solium*, in its 2003 agenda. Cysticercosis is the single most common cause of late onset seizures in the developing world (Garcia *et al.*, 2003a). *Taenia solium* persists mainly in rural areas because of the coexistence of poor sanitation and domestic pig husbandry (thus pigs have access to raw human sewage or faeces). In developed countries cysticercosis was eradicated by improving sanitation and controlling domestic pig raising. In developing countries, however, pig husbandry methods which allow the transmission cycle of *Taenia solium* to persist are largely economically motivated. Free ranging pigs minimize or cancel feed investment. Moreover, surveillance of pigs at point of slaughter, which is the current standard of control, relies on inspection systems being enforced and adhered to. As infected carcasses are confiscated, thereby depriving smallholder farmers of an income, most pigs are killed clandestinely and infected pork remains widely available (Gonzales, 2003). This thesis examined the risk factors associated with neurocysticercosis and taeniasis in a population of epileptic patients in Busia District, Kenya. Questionnaires on household information, pig husbandry practices, hygiene and santitation, presence of epileptics and tapeworm carriers in the household as well as knowledge and awareness regarding tapeworm infection and cysticercosis were administered to the population of epileptics. Serum was collected from the patients and three diagnostic tests performed to ascertain the prevalence of *Taenia solium* infection. Chapter 4 of this thesis presents the results of a univariate and logistic regression multivariate analysis of associated risk factors and prevalence rates by serological testing using a Bayesian approach.

5.2 Univariate Analysis

The variables to emerge from the univariate analysis as significantly increasing or decreasing the risk of association with seropositivity which were in line with the causal model and predicted risk factors were:

Latrine used	Decreased	Yes
History of head injury	Decreased	Yes
Had skin nodules in the past	Increase	Yes
Farmer	Increase	Yes
Other epileptics in household	Increase	Yes
Latrine not used (observation)	Increase	Yes
Has seen nodules in pigs	Increase	Yes
Very poor	Increase	Yes

5.2.1 Latrine used or not used

Using a latrine breaks the cycle of transmission for *Taenia solium* cysticercosis. This finding is in keeping with studies carried out in China (Cao *et al.*, 1996) and Mexico, where infection rates of porcine cysticercosis and were habitually higher in pigs that had access to latrines or were fed human faeces (Sarti *et al.*, 1992). In this study, not using a latrine is significantly associated with seropositivity (OR = 2.1, P = 0.045) as using a latrine prevents pigs having access to human faeces potentially contaminated with tapeworm eggs and therefore does not allow the cycle of transmission to continue.

5.2.2 History of head injury

A history of head injury could signify epilepsy acquired by means other than *Taenia solium* tapeworm. Epilepsy can also occur after a significant head trauma as well as meningitis.

5.2.3 Had skin nodules in the past

Seeing nodules under the skin is also significantly associated (OR = 10.6, *P* = 0.008) and this is consistent with greater logical risk as having a tapeworm infection in the past is closely linked to the potential for acquiring neurocysticercosis. The presence of nodules under the skin could indicate a past or ongoing infection with cysts. In a study conducted in India, one hundred and tweny patients presenting with subcutaneous swellings, were diagnosed as cysticercus or suspicious of parasitic inflammation (Uma *et al.*, 2008). In Burkina Faso, six cases were studied from 3 men

and two women, ranging from 25 to 57 years old. Three of them had neurological complications as convulsions and headaches. The nodules were painful in one case (Barro-Traore *et al.*, 2008).

5.2.4 Farmer

Raising pigs has a direct association with an increase in risk for seropositivity for cysticercosis. In this study, a positive test result by Ab ELISA was significantly associated with being a farmer (OR = 3.7, P = 0.012). This is to be expected as farmers would have more exposure to soil contaminated with tapeworm eggs. The cycle of transmission is perpetuated as long as humans and pigs live in close proximity. In China, in a population-based case-control study of rural respondents in Shandong Province, those who raised pigs had an almost 3 fold greater association with seropositivity, a relationship which was statistically significant (OR = 2.6, CI = 1.2-5.7; $P < 0.05$) (Cao, 1997). In Peru, raising pigs, irrespective of the number, was associated with being seropositive (232/1,562, 14.9% versus 123/1,021, 12.0%; OR 1.27; CI 1.01, 1.61; $P < 0.043$) (Garcia *et al.*, 1995; Garcia *et al.*, 2003b). In India, where it was found that 78% of children of pig farmers passed taeniid eggs in their stools (Banerjee *et al.*, 1994), having no separate place to keep pigs apart from the household space and having epilepsy in the family were identified as risk factors for NCC clustering in a family (Prasad *et al.*, 2009).

5.2.5 Other epileptics in the household

The presence of other epileptics in the household can be interpreted as an indication that the tapeworm and therefore neurocysticercosis is present. In this study, the presence of another epileptic in the household is significant (OR = 3.6, P = 0.18) which is consistent with more people in the household being exposed to tapeworm eggs and genetic factors being less important than risk factors for epilepsy. Data from the Americas indicates that the main risk for both human neurocysticercosis and swine cysticercosis is the presence of a tapeworm carrier in the household (Flisser *et al.*, 2003). Other studies from Bolivia, Ecuador and Guatemala indicate this same association (Cruz *et al.*, 1989; Garcia-Noval *et al.*, 1996; Bern *et al.*, 1999).

5.2.6 Nodules seen in pigs

The failure of people to recognize the presence of cysts in pigs as being positively associated with cysticercosis morbidity contributes to the lack of control of the disease. In a study in Zambia, observation of cysts by farmers was high in all districts, but did not deter them from eating or selling pork. Most of the respondents (83.3%) had observed cysts in pork. Of the respondents that observed cysts in pork, 20.1% ate and 18.3% sold the meat (Sikasunge *et al.*, 2007). In Tanzania, farmers regularly slaughter pigs clandestinely and consume the meat themselves or sell to a neighbour, regardless of whether it is contaminated with cysts (Boa *et al.*, 2006). In this study, patients had almost a 2 fold greater odds of being seropositive for cysticercosis if they had seen subcutaneous nodules in pigs (OR = 1.7, 0.92-3.34).

5.3 Prevalence findings

The prevalence rate in this study is lower than anticipated. Compared to studies in other parts of Africa with similar agro-ecological studies such as a study done in Burundi examining the correlation in seropositivity between the Ab ELISA and the Ag ELISA which showed 40.5% and a 26.1% positive result respectively, the prevalence rate found in this study of 0.38% when using a Bayesian approach for three tests is very low. One explanation for this is that it is possible that in this population of epileptics NCC was not a major cause of epilepsy and that other factors are responsible. If this is the case, it would be important to investigate risk mitigating practices that would explain why although many of the risk factors are present the prevalence of NCC is low. However, other common causes of epilepsy (genetics, infection, trauma) were not sufficient to explain more thana minority of cases and other studies suggested cysticercosis might be a problem, given the prevalence of porcine cysticercosis in the District (Githigia, 2000; Mutua *et al.*, 2006). It is also possible that the test results were not indicative of NCC as the Ab ELISA in particular is known to cross react with other helminthic infections. As Dorny suggests, immunodiagnosis by serology must be used in conjunction with neuro-imaging techniques, patient history and other neurological symptoms in order to make a definitive diagnsosis (Dorny *et al.*, 2003). The application of ELISA for the detection of circulating parasite antigens may present some diagnostic advantages since it demonstrates not only exposure but also active infections. Only a few of the

current techniques have been standardised and fully validated, making comparisons between studies difficult. In surveys on cysticercosis, antibody detection systems have been useful in identifying the risk factors associated with transmission of *Taenia solium* and a high seroprevalence in a community can indicate a "hot spot" and the need to look further. The potential use of immunodiagnostic tools to identify cases of neurocysticercosis (NCC) in man is subject to debate. The correlation between a positive serology and neurological symptoms and/or lesions indicative for NCC on neuro-imaging techniques is poor to fair in most studies. This may be explained by the unpredictable clinical outcome of the infection and the variable immunological response of the human host to infection (Dorny *et al.*, 2003).

In this study, the serological results would have ideally been compared with a CT scan to assist in validating the diagnostic techniques. Although this was originally going to be part of this study, political problems in the country prevented this from taking place. It would be interesting to compare serological and neuro-imaging results at some point in the future as these neuro-imaging methods are available, unlike in some developing countries where they are inaccessible and/or too expensive.

5.4 Poverty findings

Although seropositive epileptics scored higher on all 5 indices of poverty, this association was not significant. Cysticercosis is considered a disease of poverty and this was not in accordance with our initial hypothesis. Given that Busia District is one of the poorest in Kenya (CBS, 2003), it is possible that poverty is so prevalent that the association between poverty, risk factors and seroprevalence was not significant. Again, further studies to disaggregate poverty further and examine seropositivity and a rural/urban correlation may prove useful in looking at the role poverty plays in cysticercosis.

5.5 Multivariate risk factor analysis

The multivariate analysis which took into account confounding risk factors revealed that only four factors were significant. These were, no other epileptics living in the

household, the respondent living in Nambale Division, seeing skin nodules more than one year ago and farming as the principal occupation of the household.

test	*Odds Ratio*	*Std. Err.*	*P*	*95% Conf.*	*Interval*
No other epileptics in household	0.22	0.13	0.01	0.07	0.68
Lives in Nambale	12.32	14.91	0.04	1.15	131.95
Skin nodules > 1 year ago	13.04	16.79	0.05	1.05	162.55
Farming	4.58	3.62	0.05	0.97	21.58

5.5.1 Lack of other epileptics in the household

This risk factor proved to be protective which is in keeping with other studies previously cited indicating that having an epileptic in the household increases the risk of association with seropositivity.

5.5.2 Lives in Nambale

Cysticercosis cases tend to cluster as transmission is related to hygiene and santiation and other risk factors which can be prevalent throughout a community. Several studies have shown that high seropositivity rates for cysticercosis are significantly associated with tapeworm carrier clusters and that seropositive persons are significantly clustered within households, particularly, in households in which a member reported a history of having passed tapeworm proglottids, as well as with individuals with a clinical history of seizures (Diaz-Camacho *et al.*, 1990, Sarti *et al.*, 1992).

5.5.3 Skin Nodules

This risk factor is consistent with that found in the univariate analysis.

5.5.4 Farming

This risk factor is consistent with that found in the univariate analysis.

5.6 Conclusion

Although the prevalence of cysticercosis was low in this study, many of the risk factors examined and found to be significant are comparable to those found in other studies around the world. Cysticercosis has been found to be an emerging problem in other parts of Africa (Mafojane *et al.*, 2003; Zoli *et al.*, 2003a), and it stands to

reason that given the relative similarities between these places and Busia District, it will become a problem soon. It would be interesting to follow up with further studies to examine the validity of the diagnostics used and the influence of genetic factors on the prevalence of epilepsy in the district, always keeping in mind the societal context and sensitivities of culture when carrying out research.

5.7 Importance of Societal Context in Study

Many factors exist within a society with the potential to provide bias in enumeration and to influence the researcher when drawing conclusions. These can include general characteristics which when known about a particular culture, are helpful when formulating questionnaires or analysing information from questions asked. Examples of this kind of knowledge include knowing when asking whether meat is "cooked", whether in this culture people generally eat meat rare or well cooked. If people never eat rare meat, then "cooked" can be taken to mean well cooked. Another example where general knowledge of a culture is useful is knowing peoples' general demeanour- whether aggressive, confrontational or placatory. In a culture such as that in Kenya, people generally want to be affirmative in their answers. This tendency can arise from a variety of reasons; preserving the general harmony of a society without confrontation, appearing knowledgeable is better than appearing ignorant or that being helpful is considered a much better attribute than not. This characteristic, however, can create difficulties for the researcher in that people will answer in the affirmative whether or not they actually know what is being asked for. Another example where the placatory tendency is illustrated is in the provision of directions when being asked. In the rural areas of Kenya, it is considered impolite not to be able to provide directions for a visitor, regardless of whether one knows where the place being asked of actually is. It is considered much better to provide inaccurate and often extremely unhelpful directions which do not assist in actually reaching the desired destination, rather than to provide none at all. The researcher must have a certain depth of knowledge of the culture in which the research is being carried out in order to bring a more accurate interpretation to the planning and results of the work.

5.8 Opportunities for Further Research

Given that CT scans were not able to be administered to any study participants, and that different respondents were administered different parts of the study at different times, opportunities to explore further the accuracy of the diagnostic tests and potential correlation between risk factors and seropositivity exist. Follow-up to this study should include the following:

- Serological testing of all three diagnostic tests given to all those respondents who answered the risk factor questionnaire
- CT scans given to all those respondents with a positive result from any of the three tests
- Risk factor questionnaires administered to those respondents who have already provided serum for testing.

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Appendix A: Ethical Approval

KENYA MEDICAL RESEARCH INSTITUTE

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Tel: +254 (020) 2722541, 2713349, 0722-205901, 0733-400003, Fax +254 (020) 2720030,
E-mail: director@kemri.org; info@kemri.org Website: www.kemri.org

KEMRI/RES/7/3/1 **December 8, 2006**

Ms. K Downie-Ngini
ILRI,
NAIROBI

Dear Madam,

Re: **NON-SSC - Assessing** the Burden of *Taenia solium* Neurocystercercosis: A case study of Busia District, Kenya, **by K Downie-Ngini *et al* (ILRI)**

Thank you for timely response dated 7th December 2006.

We acknowledge receipt of the revised protocol and informed consent form (ICF). We note the change in the title to read "**Assessing the Burden of *Taenia solium* Neurocystercercosis: A case study of Busia District, Kenya** "and the change in the ICF to indicate that 5cc of blood will be drawn and sent to Belgium for serology testing. Due consideration has been given to ethical issues and the study is granted approval for a period of one year with effect from December 8 2006 to December 7, 2007.

You may proceed with your study. You are responsible for reporting to the Ethical Review Committee any changes to the protocol or in the Informed Consent Document. This includes changes to research design or procedures that could introduce new or more than minimum risk to human subjects.

Yours faithfully,

R.C. Kithinji,
For: Secretary,
KEMRI/NATIONAL ETHICAL REVIEW COMMITTEE

Appendix B: CWGESA Questionnaire

TAENIOSIS/CYSTICERCOSIS QUESTIONNAIRE

Last name : _____ First Name : _____

If Child then : Questionnaire number : _____

Father's Name :_____ District _____

Division: _____

Mother's Name:_____ Location: _____

Sub-location: _____

Village _____

Hut (house) number _____

How long have you lived in this village?____ (yrs.)

GPS Reading North:_____(Format N00.xxxxx)

East:: _____(Format E00.xxxxx)

Altitude: _____(Format xxxx m)

1 How old are you? _____ (years)

2 What is your date of birth? ____ Day ____ Month _____ Year

3 Sex ☐ Male ☐ Female

4 What is the highest schooling grade you have completed?

☐ None ☐ Primary school

☐ Middle School ☐ High school

5 What further education have you completed?

☐ None ☐ College

☐ University ☐ Technical/Vocational

6 What is your occupation? _____

7 How many days of work have you missed because of illness in the past month? _____ days

8 How many days of work have you missed because of illness in the past year? _____ days

9 Where do you usually get your drinking water?

☐ River ☐ Bore-hole

☐ Well ☐ Other (please specify) _____

10 Do you boil your drinking water?

☐ Always ☐ Almost always

☐ Sometimes ☐ Never

11 How often do you eat pork?

☐ At least once a month ☐ Less than once a month but at least once a year

☐ Less than once a year ☐ Never

12 How is the pork that you eat prepared? *Check all that apply.*]

☐ Boiling ☐ Barbeque

☐ Fried ☐ Others

(specify)_____

13 Do you have a latrine at home?

☐ Yes ☐ No (Skip to Q 14)

13.1 How often do you use a latrine when you have to defecate?

☐ Always ☐ Sometimes ☐ Never

14 Do you keep pigs?

☐ Yes ☐ No (Skip to Q 15)

14.1 What type of pigs do you keep?

☐ Foreign ☐ Native

☐ Both foreign and native ☐ Can not remember, do not know

14.2 Of the pigs that you have, how many are for? [*read each choice and record the number*]

Home consumption _____ Trading _____

Reproduction _____ Other (specify): _____

14.3 How do you keep your pigs… [*read questions 14.3.1 to 14.3.4 one after the other*]

14.3.1 During the planting season

☐ In a pen ☐ Free ranged

☐ Tethering ☐ Other (specify): _____

14.3.2 During the growing season

☐ In a pen ☐ Free ranged

☐ Tethering ☐ Other (specify): _____

14.3.3 During the harvesting season

☐ In a pen ☐ Free ranged

☐ Tethering ☐ Other (specify): _____

14.3.4 During the fallowing season

☐ In a pen ☐ Free ranged

☐ Tethering ☐ Other (specify): _____

14.4 What do your pigs eat? [*Check all that apply.*]

☐ Pasture ☐ Kitchen left overs

☐ Commercial feeds ☐ Other (specify): _____

14.5 How often do you slaughter pigs at home?

☐ At least once a month ☐ Less than once a month but at least once a year

☐ Less than once a year

☐ Never (Skip to Q 14.6) ☐ Can not remember, do not know (Skip to Q 14.6)

14.5.1 If ever, how often was the meat inspected by a meat inspector?

☐ Always ☐ Almost always

☐ Sometimes ☐ Never

☐ Can not remember, do not know

14.6 . What price do you usually sell your pigs when they are ready to be slaughtered (specify the currency used, this can be money or barter)? _____

_____

14.7. What price do you usually sell your piglets (aged 4 months or less) (specify the currency used, this can be money or barter)? _____

_____ (SKIP TO Q 16)

15. Have you ever owned pigs? *[If they answer "yes", ask when they owned pigs]*

☐ Yes, in the past year ☐ Yes, one (1) to five (5) years ago

☐ Yes, more than five (5) years ago

☐ No (skip to Q 17)

15.1. What kind of pigs were they?

☐ Foreign ☐ Native

☐ Both foreign and native ☐ Can not remember, do not know

16. Were you ever told that your pigs or piglets were infected with cysts (cysticercosis)?

☐ Yes ☐ No (Skip to Q 17)

16.1. When were you told that your pig or piglets were infected with cysts (cysticercosis)?

☐ In the past year ☐ One (1) to five (5) years ago

☐ More than five (5) years ago

☐ Never told (skip to Q 17) ☐ Can not remember, do not know (skip to Q 17)

16.1.1 When that happened, were you able to sell your pig(s) or piglets?

☐ Sold both ☐ Sold pigs but not piglets

☐ Sold piglets but not pigs (skip to Q 16.1.3)

☐ Could not sell either (skip to Q 17)

☐ Can not remember, do not know (skip to Q 17)

16.1.2 When that happened, what price did you sell your pigs (aged more than 4 months) (specify the currency used, this can be money or barter)? _____

_____

16.1.3 When that happened, what price did you sell your piglets (aged 4 months or less) (specify the currency used, this can be money or barter)? _____

_____

17. Have you ever seen or heard of white nodules (rice) in pig carcasses?

☐ Yes ☐ No (Skip to 18)

17.1 Where can you find nodules on a live pig?

☐ It is not possible to find them on a live pig

☐ Under the skin ☐ Under the tongue

☐ I don't know ☐ Somewhere else (Specify) _____

17.2 How do pigs get these nodules?

☐ By eating human faeces ☐ By eating pig faeces

☐ From another infected pig ☐ Other (Specify) _____

☐ I don't know

17.3 What would you do if you discovered that your pig had nodules?

☐ Sell the pig ☐ Treat it with herbs

☐ Pierce the nodules ☐ Other (Specify) _____

☐ I don't know

18. Have you ever heard of tapeworm infection in humans?

☐ Yes ☐ No (Skip to question 19)

18.1 How did you learn about it?

☐ By a doctor ☐ By a friend or family member

☐ By a traditional healer ☐ On the radio / newspaper

☐ Other (Specify) _____

18.2 How does a person know if they have a tapeworm?

☐ They can see it in their faeces ☐ They have diarrhea

☐ They have fever ☐ Other (Specify)

_____

☐ I don't know

18.3 Have you ever had a tapeworm or seen small parts (segments) of worms in your faeces? (*Show photographs of proglottids*)

☐ Yes ☐ No (SKIP TO Q 18.4)

☐ I don't know/can not remember (SKIP TO Q 18.4)

18.3.1 When that happened, what did you do? [*check all that applies*]

☐ Went to a primary health care provider (hospital, clinic, dispensary)

☐ Went to the pharmacy to get a drug to treat it

☐ Went to a traditional healer ☐ Did nothing

☐ I can not remember, I do not know

18.4 How does a person get tapeworm infection?

☐ They do not wash their hands ☐ They eat undercooked pig meat

☐ They are in contact with an infected person ☐ Other (Specify) _____

☐ I don't know

19. Have you ever had skin nodules or hard lumps under the skin? (*Show photograph of person with subcutaneous cysticercosis nodules*)

☐ Yes, currently has ☐ Yes in the past year, but not currently

☐ Yes, one year or more ago, but not currently ☐ No

☐ Can not remember, do not know

20. Have you ever had bad headaches that lasted more than a few days?

☐ Yes, currently has ☐ Yes in the past year, but not currently

☐ Yes, one year or more ago, but not currently ☐ No

☐ Can not remember, do not know

21. Have you ever had any of the following?

21.1 Sudden loss of consciousness and episodes of incontinence or foaming of the mouth or tongue biting?

☐ Yes, currently has ☐ Yes in the past year, but not currently

☐ Yes, one year or more ago, but not currently

☐ No (Skip to Q 21.2) ☐ Can not remember, do not know (Skip to Q 21.2)

21.1.1 (If yes) How often has this happened?

☐ Only once ☐ More than once

21.1.2 How old were you when this first happened? _____ years

21.2 A brief period of absence(s) or loss(es) of contact with the surroundings that starts suddenly?

☐ Yes, currently has ☐ Yes in the past year, but not currently

☐ Yes, one year or more ago, but not currently

☐ No (Skip to Q 21.3) ☐ Can not remember, do not know (Skip to Q 21.3)

21.2.1 How often has this happened?

☐ Only once ☐ More than once

21.2.2 How old were you when this first happened? _____ years

21.3 Uncontrollable twitching or jerking or abnormal movements of one or more limb(s) (convulsions) that starts suddenly and lasts for a period of a few minutes?

☐ Yes, currently has ☐ Yes in the past year, but not currently

☐ Yes, one year or more ago, but not currently

☐ No (Skip to Q 21.4) ☐ Can not remember, do not know (Skip to Q 21.4)

21.3.1 How often has this happened?

☐ Only once ☐ More than once

21.3.2 How old were you when this first happened? _____ years

21.4 Sudden onset of a brief period of hearing or smelling or seeing things that are not there or feeling strange body sensations?

☐ Yes, currently has ☐ Yes in the past year, but not currently

☐ Yes, one year or more ago, but not currently

☐ No (Skip to Q 21.5) ☐ Can not remember, do not know (Skip to Q 21.5)

21.4.1 How often has this happened?

☐ Only once ☐ More than once

21.4.2 How old were you when this first happened? _____ years

21.5 Were you ever told that you had epilepsy or that you had had an epileptic seizure?

☐ Yes, currently has ☐ Yes in the past year, but not currently

☐ Yes, one year or more ago, but not currently ☐ No

☐ Can not remember, do not know

21.6 Have you ever had seizures or fits?

☐ Yes, currently has ☐ Yes in the past year, but not currently

☐ Yes, one year or more ago, but not currently

☐ No (skip to Q 22) ☐ Can not remember, do not know (skip to Q 22)

21.6.1 How often has this happened?

☐ Only once ☐ More than once

21.6.2 How old were you when this first happened? _____ years

[If the interviewee has answered "no" to questions 21.1-21.6, the interview is finished. Go to last page and complete questions 30 & 31 based on observation.]

THANK YOU VERY MUCH FOR YOUR COOPERATION

[Otherwise, please continue with the questionnaire]

[Interviewer: If they answered "yes" to any of the questions 21.1-21.6, ask the following, otherwise, SKIP to Q. 25.]

22. Have you had any of the following?

22.1 Head injury that made you lose consciousness? ☐ Yes ☐ No (skip to Q 22.2)

22.1.1 If yes, when did your seizure symptoms start?

☐ Before head injury ☐ Soon after head injury

22.2 Meningitis (brain infection) during childhood? ☐ Yes ☐ No

22.2.1 If yes, when did your seizure symptoms start?

☐ Before an attack of meningitis ☐ Soon after an attack of meningitis

23. What happens to you when you have a seizure or a fit? _____

_____

24. Have you ever hurt yourself when you lose consciousness or during a seizure?

☐ Yes ☐ No

☐ I do not lose consciousness or have seizures (skip to question Q 25)

☐ Cannot remember (skip to question Q 25)

24.1 If yes, how did you hurt yourself?

☐ Fell in the fire ☐ Fell in the water

☐ Fell off your bicycle ☐ Fell while walking along the road

☐ Cut yourself ☐ Other (specify) _____

25. Is there someone in your household with epilepsy or seizures?

☐ Yes, currently is ☐ Yes in the past year, but not currently

☐ Yes, one year or more ago, but not currently ☐ No

25.1 (If yes) Who in your household has epilepsy or seizures? [*check all that apply*]

☐ Mother ☐ Father

☐ Brother/sister ☐ Child

☐ Other relative ☐ Other (specify) _____

(Interviewer: Read the following statement)

Now I want to ask you a few questions about your treatments for *[insert name of symptom or condition they reported having in question 21.1-21.6]*

26. Have you ever consulted a health provider because of this condition?

☐ No (skip to Q 27) ☐ Cannot remember (skip to Q 27)

☐ Yes

26.2 When was the <u>last</u> time you consulted a health provider for your condition?

☐ Within the past month ☐ Within the past year

☐ From one (1) to five (5) years ago ☐ More than five (5) years ago

☐ Can not remember, not sure

26.3 What kind of health provider(s) did you consult and how many times in the past 5 years [*check several boxes if appropriate*]?

☐ A physician _____ times ☐ A neurologist _____ times

☐ A nurse _____ times ☐ A traditional healer _____ times

☐ A psychiatrist/psychologist _____ times

☐ Other (specify _____) _____ times

☐ Can not remember, not sure

26.4 How much did it cost each time you consulted with one health provider [*specify the currency used*]?

☐ A physician _____ ☐ A neurologist _____

☐ A nurse _____ ☐ A traditional healer _____

☐ A psychiatrist / psychologist _____

☐ Other (specify _____) _____

☐ Can not remember, not sure

26.5 How far is the health provider from your house and how did you get there (foot, bicycle, bus, train, taxi, car)?

☐ Physician at _____ km reached by_____ ☐ Neurologist at _____ km reached by____

☐ Nurse at ____ km reached by____ ☐ Traditional healer at ___ km reached by___

☐ Other (specify _____) at _____ km reached by_____

☐ Can not remember

27. Have you ever been hospitalised because of this condition?

☐ No (skip to Q 28) ☐ Cannot remember (skip to Q 28)

☐ Yes Name of Facility _____

27.2 How many times have you been hospitalised in the past 5 years? _____times

27.3 When were you <u>last</u> hospitalised? _____(month) _____(year)

27.3.1 How many days did you stay in hospital? _____ (days)

27.3.2 How much did it cost (specify the currency) _____

27.3.3 How far is the hospital from your house? _____ km

27.3.4 How did you get to the hospital?

☐ By foot/ bicycle ☐ By bus ☐ By taxi

☐ By car ☐ By train ☐ Other (specify)

_____

28. Did you ever have any medical tests because of this condition?

☐ No (skip to Q 29) ☐ Cannot remember, do not know (skip to Q 29)

☐ Yes

28.2 What kind of test was it (check as many boxes as appropriate)?

☐ Blood test for cysticercosis ☐ CT scan of the brain

☐ X-Ray of the brain ☐ MRI of the brain

☐ Electroencephalogram (EEG) ☐ Other (please specify) _____

☐ Can not remember, not sure

28.3 When was the last time you had a medical test for this condition?

☐ Within the past month ☐ Within the past year

☐ From one (1) to five (5) years ago ☐ More than five (5) years ago

☐ Can not remember, not sure

28.4 How much did it cost for each test (specify the currency used)?

☐ Blood test for cysticercosis_____ ☐ CT scan of the brain _____

☐ X-Ray of the brain_____ ☐ MRI of the brain _____

☐ Electroencephalogram _____ ☐ Other (specify) _____

☐ Can not remember, not sure

28.5 How far from your house did you have to travel for this test and how did you get there (foot, bicycle, bus, train, taxi, car)?

☐ Blood test for cysticercosis at _____ km at (Facility Name)_____reached by_____

☐ CT scan at _____ km at (Facility Name)_____reached by_____

☐ X-Ray at _____ km at (Facility Name)_____reached by_____

☐ MRI at _____ km at (Facility Name)_____reached by_____

☐ Electroencephalogram at _____ km at (Facility Name)_____reached by_____

☐ Other (specify _____) at _____ km at (Facility Name) _____ reached by_____

☐ Can not remember, not sure

29. Were you ever treated for this condition?

☐ No (the interview is finished) ☐ Can't remember, do not know (interview is finished)

☐ Yes

29.2 When was the last time you used medication for your condition?

☐ Within the past month ☐ Within the past year

☐ From one (1) to five (5) years ago ☐ More than five (5) years ago

☐ Can not remember, not sure

29.3 What medication was it and how many times in the past year did you have to use some (check several boxes if appropriate)?

☐ Phenobarbital _____ times

☐ Dilantin/Tegritol/ Phentoin Sodium _____ times (tick box and underline specific drug name)

☐ Valproic acid _____ times ☐ Traditional medicine _____ times

☐ Other (specify _____) _____ times

☐ Can not remember, not sure

29.4 How much did it cost each time you bought this medication (specify the currency used)?

☐ Phenobarbital _____

☐ Dilantin/Tegritol/Phentoin Sodium _____ (tick box and underline specific drug name)

☐ Valproic acid _____ ☐ Traditional medicine _____

☐ Received for free from health care provider (I did not pay for it myself) _____

☐ Other (specify _____)

☐ Cannot remember, not sure

The following two items should be completed for ALL respondents after direct observation of latrine.

30. Presence and type of latrine (**to be assessed by direct observation)**:

☐ Absent ☐ Present and completely enclosed

☐ Present and partially enclosed ☐ Present and open (easily accessible to roaming pigs)

31. Is there evidence of recent use of the latrine (by anyone) (**to be assessed by direct observation**)?:

☐ Yes ☐ No

THIS IS THE END OF THE INTERVIEW

THANK YOU VERY MUCH FOR YOUR COOPERATION.

INTERVIEWER: _____

DATE OF INTERVIEW: _____

Appendix C: Univariate Analysis – Full List of Indicators

	Cases			Controls					
Exposure	Total	Exposed	%	Total	Exposed	%	Odds	Ratio	p
dx_tw_fever	**81**	**9**	**11.11**	**129**	**1**	**0.78**	**15.48**	**[2.01-686.05]**	**0.001**
lat_used_ob	**81**	**39**	**48.15**	**129**	**86**	**66.67**	**0.38**	**[0.18-0.81]**	**0.006**
skn_nd_mr_1_	**81**	**6**	**7.41**	**129**	**1**	**0.78**	**10.59**	**[1.23-490.90]**	**0.008**
farming	**81**	**11**	**13.58**	**129**	**6**	**4.65**	**3.67**	**[1.14-12.85]**	**0.012**
other_epi_hh	**81**	**10**	**12.35**	**129**	**5**	**3.88**	**3.6**	**[1.05-13.97]**	**0.018**
boil_all	**81**	**13**	**16.05**	**129**	**8**	**6.2**	**2.92**	**[1.05-8.54]**	**0.02**
prkyrly	**81**	**9**	**11.11**	**129**	**4**	**3.1**	**3.83**	**[1.01-17.60]**	**0.022**
get_tw_eat_p	**81**	**17**	**20.99**	**129**	**14**	**10.85**	**2.39**	**[0.98-5.86]**	**0.033**
head_inj	**81**	**13**	**16.05**	**129**	**37**	**28.68**	**0.47**	**[0.21-1.01]**	**0.037**
seizure_lst_	**81**	**12**	**14.81**	**129**	**8**	**6.2**	**2.63**	**[0.93-7.79]**	**0.039**
lat_not_usd	**81**	**20**	**24.69**	**129**	**17**	**13.18**	**2.08**	**[0.95-4.59]**	**0.045**
boil_nev	**81**	**56**	**69.14**	**129**	**103**	**79.84**	**0.52**	**[0.24-1.10]**	**0.06**
boil_all_som	**81**	**21**	**25.93**	**129**	**20**	**15.5**	**1.93**	**[0.91-4.10]**	**0.06**
seen_nod_pg	**81**	**33**	**40.74**	**129**	**38**	**29.46**	**1.76**	**[0.92-3.34]**	**0.063**
feed_comm	**81**	**3**	**3.7**	**129**	**1**	**0.78**	**6.71**	**[0.48-361.00]**	**0.072**
feed_other	**81**	**3**	**3.7**	**129**	**1**	**0.78**	**6.71**	**[0.48-361.00]**	**0.072**
drnk_river	**81**	**20**	**24.69**	**129**	**47**	**36.43**	**0.57**	**[0.29-1.10]**	**0.074**
epi_lst_yr	**81**	**8**	**9.88**	**129**	**5**	**3.88**	**2.75**	**[0.75-11.06]**	**0.077**
nambale	**81**	**8**	**9.88**	**129**	**5**	**3.88**	**2.72**	**[0.75-10.92]**	**0.079**
pv_ind_di	**81**	**23**	**28.4**	**129**	**24**	**18.6**	**1.78**	**[0.87-3.62]**	**0.085**
see_smell_du	**81**	**4**	**4.94**	**129**	**15**	**11.63**	**0.38**	**[0.09-1.27]**	**0.088**
lat_nev	**81**	**0**	**0**	**129**	**4**	**3.1**	**0**	**[0.00-1.37]**	**0.093**
twitch_strt_	81	0	0	129	0	0	2.83	[0.64-14.16]	0.105
if_cyst_cut	81	4	4.94	129	1	0.78	5.39	[0.48-273.84]	0.108
bothpig	81	2	2.47	129	0	0	.	[0.63-.]	0.115
native_pig	81	19	23.46	129	25	19.38	0	[0.00-1.58]	0.115
kill_yrly	81	0	0	129	4	3.1	0	[0.00-1.66]	0.122
fit_mr_1_yr	81	3	3.7	129	1	0.78	4.95	[0.39-261.69]	0.13
see_smell_no	81	55	67.9	129	73	56.59	1.61	[0.83-3.19]	0.132
age10	81	4	4.94	129	14	10.85	0.43	[0.10-1.43]	0.136
kill_nev	81	19	23.46	129	29	22.48	4.59	[0.51-217.35]	0.139
kill_any	81	1	1.23	129	7	5.43	0.22	[0.00-1.97]	0.139
for_pig	81	1	1.23	129	0	0	.	[0.00-.]	0.154
nat_pig	81	19	23.46	129	40	31.01	0	[0.00-.]	0.154
feed_kitch	81	19	23.46	129	39	30.23	0	[0.00-.]	0.159
see_smell_ls	81	4	4.94	129	2	1.55	3.22	[0.45-36.24]	0.161
fallow_pg_ro	81	6	7.41	129	20	15.5	0.45	[0.11-1.65]	0.176
drnk_well	81	17	20.99	129	18	13.95	1.65	[0.74-3.67]	0.178
feed_past	81	17	20.99	129	27	20.93	2.52	[0.55-15.69]	0.188
pgother	81	1	1.23	129	0	0	.	[0.00-.]	0.19
fit_fell_wal	81	1	1.23	129	6	4.65	0.26	[0.01-2.29]	0.192
fit_cut	81	1	1.23	129	0	0	.	[0.00-.]	0.198
hd_lst_yr	81	8	9.88	129	7	5.43	1.98	[0.60-6.70]	0.199
twitch_no	81	12	14.81	129	12	9.3	1.73	[0.67-4.49]	0.204
if_cyst_trea	81	2	2.47	129	6	4.65	0.35	[0.03-2.21]	0.205
fit_hurt_oth	81	16	19.75	129	35	27.13	0.63	[0.28-1.41]	0.221

see_smell_no	81	12	14.81	129	27	20.93	0.63	[0.27-1.41]	0.229
kept_pig_pre	81	17	20.99	129	19	14.73	1.55	[0.70-3.41]	0.235
lat_alm_al	81	0	0	129	2	1.55	0	[0.00-2.80]	0.238
see_smell_mo	81	53	65.43	129	75	58.14	0.42	[0.06-2.30]	0.242
see_smell_on	81	5	6.17	129	3	2.33	2.36	[0.43-15.74]	0.242
fit_now	81	63	77.78	129	108	83.72	0.63	[0.26-1.50]	0.242
epi_start_le	81	60	74.07	129	95	73.64	0.57	[0.19-1.66]	0.243
seizure_mr_1	81	0	0	129	2	1.55	0	[0.00-3.05]	0.259
pg_trde	81	12	14.81	129	15	11.63	1.94	[0.53-7.40]	0.26
fit_no	81	6	7.41	129	5	3.88	1.99	[0.49-8.55]	0.261
twitch_dunkn	81	0	0	129	2	1.55	0	[0.00-3.11]	0.264
pg_hm_cnsmp	81	1	1.23	129	5	3.88	0.3	[0.01-3.08]	0.267
cyst_mr_5_yr	81	1	1.23	129	0	0		[0.00-.]	0.268
keep_pig	81	20	24.69	129	41	31.78	0.7	[0.35-1.38]	0.271
prk_frq	81	51	62.96	129	82	63.57	1.91	[0.53-8.56]	0.281
pen_all	81	4	4.94	129	4	3.1	2.25	[0.36-13.55]	0.283
kill_mthly	81	0	0	129	2	1.55	0	[0.00-3.49]	0.283
seizure_now	81	59	72.84	129	101	78.29	0.68	[0.32-1.48]	0.286
prknevertrue	81	4	4.94	129	11	8.53	0.53	[0.12-1.90]	0.29
otherepiany	81	24	29.63	129	30	23.26	1.41	[0.70-2.82]	0.293
lat_all	81	62	76.54	129	85	65.89	1.7	[0.57-5.70]	0.298
hd_now	81	55	67.9	129	98	75.97	0.7	[0.34-1.47]	0.302
hd_mr_1_yr	81	3	3.7	129	2	1.55	2.52	[0.28-30.70]	0.302
abl_sel_cyst	81	0	0	129	1	0.78	0	[0.00-.]	0.302
prk_mthyr	81	60	74.07	129	86	66.67	1.55	[0.62-4.14]	0.311
grow_pg_rope	81	11	13.58	129	26	20.16	0.56	[0.16-2.01]	0.312
lrn_tw_dr	81	12	14.81	129	13	10.08	1.53	[0.60-3.88]	0.317
lrn_tw_trad	81	2	2.47	129	1	0.78	3.2	[0.16-190.31]	0.32
faint_mr_1_y	81	3	3.7	129	2	1.55	2.4	[0.27-29.28]	0.329
drinkother	81	16	19.75	129	33	25.58	0.72	[0.34-1.48]	0.333
all_free	81	5	6.17	129	6	4.65	1.89	[0.39-8.70]	0.345
dx_tw_diar	81	10	12.35	129	10	7.75	1.57	[0.54-4.54]	0.347
skn_nod_now	81	6	7.41	129	15	11.63	0.62	[0.19-1.81]	0.349
skn_nod_lst_	81	4	4.94	129	11	8.53	0.57	[0.13-2.03]	0.349
plant_pg_rop	81	10	12.35	129	25	19.38	0.6	[0.18-2.04]	0.355
pg_reprod	81	7	8.64	129	16	12.4	0.58	[0.15-2.14]	0.361
told_cysts	81	8	9.88	129	8	6.2	1.63	[0.46-5.68]	0.386
if_cyst_othe	81	1	1.23	129	3	2.33	0.37	[0.01-5.04]	0.388
fallow_pg_pe	81	4	4.94	129	5	3.88	1.89	[0.32-10.17]	0.388
no_epi_hh	81	44	54.32	129	78	60.47	0.76	[0.39-1.50]	0.39
epi_mr_1_yr	81	1	1.23	129	4	3.1	0.39	[0.01-4.08]	0.39
epi_dunknow	81	1	1.23	129	4	3.1	0.39	[0.01-4.08]	0.39
grow_pg_free	81	5	6.17	129	6	4.65	1.78	[0.36-8.22]	0.395
nod_ex_other	81	5	6.17	129	4	3.1	1.85	[0.35-10.35]	0.395
harv_pg_rope	81	9	11.11	129	22	17.05	0.63	[0.19-2.14]	0.406
prk_bby	81	0	0	129	1	0.78	0	[0.00-.]	0.41
lrn_tw_schoo	81	3	3.7	129	8	6.2	0.57	[0.10-2.49]	0.416
other	81	14	17.28	129	28	21.71	0.73	[0.31-1.66]	0.418
latrine	81	66	81.48	129	100	77.52	1.38	[0.60-3.35]	0.419
get_tw_other	81	17	20.99	129	33	25.58	0.74	[0.34-1.62]	0.422
faint_dunkno	81	0	0	129	1	0.78	0	[0.00-.]	0.423
see_tw_trad	81	0	0	129	1	0.78	0	[0.00-.]	0.427

see_tw_dk	81	0	0	129	1	0.78	0	[0.00-.]	0.427
fit_dunknwo	81	0	0	129	1	0.78	0	[0.00-.]	0.428
school	81	0	0	129	1	0.78	0	[0.00-.]	0.432
drnk_bore	81	33	40.74	129	46	35.66	1.26	[0.67-2.33]	0.442
fallow_pg_fr	81	8	9.88	129	13	10.08	1.54	[0.41-5.58]	0.46
faint_now	81	65	80.25	129	106	82.17	0.72	[0.28-1.91]	0.462
ad_onset_ep	81	13	16.05	129	15	11.63	1.35	[0.55-3.30]	0.465
harv_pg_pen	81	4	4.94	129	5	3.88	1.7	[0.29-9.05]	0.468
seen_tw_fece	81	27	33.33	129	36	27.91	1.27	[0.62-2.59]	0.473
econom_vpoor	81	46	56.79	129	75	58.14	0.79	[0.40-1.60]	0.48
age20to29	81	21	25.93	129	28	21.71	1.26	[0.62-2.53]	0.482
budalangi	81	18	22.22	129	34	26.36	0.8	[0.39-1.60]	0.499
lat_som_nev	81	6	7.41	129	12	9.3	0.7	[0.20-2.16]	0.5
lrn_tw_oth	81	4	4.94	129	4	3.1	1.6	[0.29-8.86]	0.51
if_cyst_vet	81	4	4.94	129	3	2.33	1.68	[0.25-12.54]	0.52
plant_pg_fre	81	6	7.41	129	9	6.98	1.48	[0.36-5.73]	0.527
skn_nod_dunk	81	3	3.7	129	3	2.33	1.66	[0.22-12.71]	0.537
nod_not_pos_	81	1	1.23	129	2	1.55	0.48	[0.01-9.86]	0.554
hurt_when_fi	81	46	56.79	129	76	58.91	0.83	[0.44-1.60]	0.557
if_cyst_	81	13	16.05	129	13	10.08	1.36	[0.43-4.31]	0.562
heard_hum_ta	81	60	74.07	129	90	69.77	1.22	[0.59-2.56]	0.571
fit_fell_fir	81	13	16.05	129	18	13.95	1.26	[0.50-3.10]	0.576
more_1_seiz	81	64	79.01	129	98	75.97	0.75	[0.22-2.55]	0.588
only_one_sei	81	7	8.64	129	8	6.2	1.34	[0.39-4.45]	0.588
faint_more_o	81	66	81.48	129	105	81.4	0.73	[0.20-2.77]	0.59
faint_once	81	6	7.41	129	7	5.43	1.36	[0.36-4.96]	0.59
prk_mthly	81	51	62.96	129	82	63.57	0.83	[0.39-1.80]	0.602
hd_dunk0w	81	2	2.47	129	5	3.88	0.65	[0.06-4.08]	0.605
age30up	81	14	17.28	129	26	20.16	0.83	[0.37-1.79]	0.606
lat_clsd_ob	81	28	34.57	129	47	36.43	0.86	[0.45-1.62]	0.61
twitch_lst_y	81	5	6.17	129	6	4.65	1.37	[0.32-5.61]	0.61
no_wrk	81	22	27.16	129	41	31.78	0.84	[0.40-1.77]	0.623
fit_mr_one	81	63	77.78	129	105	81.4	0.77	[0.24-2.57]	0.623
fit_one	81	7	8.64	129	9	6.98	1.3	[0.39-4.13]	0.623
plant_pg_pen	81	4	4.94	129	6	4.65	1.42	[0.26-6.95]	0.624
age_10_19	81	41	50.62	129	61	47.29	1.14	[0.63-2.07]	0.638
epi_now	81	45	55.56	129	76	58.91	0.87	[0.46-1.64]	0.641
dx_tw_dk	81	6	7.41	129	11	8.53	0.78	[0.22-2.48]	0.647
ownpig_mre_5	81	3	3.7	129	3	2.33	1.47	[0.17-12.24]	0.658
kill_less_yr	81	1	1.23	129	1	0.78	1.84	[0.02-148.65]	0.668
insp_som	81	0	0	129	1	0.78	0	[0.00-.]	0.686
insp_nev	81	1	1.23	129	6	4.65	.	[0.00-.]	0.686
grow_pg_pen	81	4	4.94	129	6	4.65	1.33	[0.24-6.56]	0.687
ed_none	81	29	35.8	129	50	38.76	0.89	[0.47-1.66]	0.693
other_epi_in	81	12	14.81	129	22	17.05	0.86	[0.36-1.97]	0.694
meningitis	81	11	13.58	129	19	14.73	0.85	[0.34-2.05]	0.703
cyst_1to5_yr	81	2	2.47	129	3	2.33	0.67	[0.04-9.11]	0.714
other_epi_in	81	4	4.94	129	8	6.2	0.8	[0.17-3.12]	0.717
busiam	81	7	8.64	129	13	10.08	0.84	[0.27-2.40]	0.73
seizure_no	81	3	3.7	129	6	4.65	0.78	[0.12-3.81]	0.735
nod_ex_pig_f	81	1	1.23	129	2	1.55	0.66	[0.01-13.44]	0.739
harv_pg_free	81	7	8.64	129	12	9.3	1.21	[0.32-4.33]	0.742

ill_after_me	81	8	9.88	129	13	10.08	0.77	[0.12-5.17]	0.745
ill_pre_men	81	4	4.94	129	5	3.88	1.3	[0.19-8.20]	0.745
lat_open_ob	81	9	11.11	129	12	9.3	1.16	[0.41-3.20]	0.745
if_cyst_sell	81	5	6.17	129	7	5.43	0.81	[0.18-3.49]	0.75
see_smell_mr	81	1	1.23	129	1	0.78	1.56	[0.02-123.48]	0.753
ss_strt_m	81	0	0	129	0	0	1.37	[0.10-19.35]	0.756
nod_ex_pig	81	2	2.47	129	2	1.55	1.38	[0.09-20.11]	0.758
owned_pig	81	36	44.44	129	54	41.86	1.09	[0.59-2.03]	0.761
see_tw_nothi	81	9	11.11	129	13	10.08	1.17	[0.36-3.62]	0.768
lrn_tw_frnd	81	36	44.44	129	54	41.86	1.09	[0.59-2.01]	0.77
nod_ex_human	81	6	7.41	129	7	5.43	1.2	[0.28-4.89]	0.772
ownpig1to5_y	81	5	6.17	129	8	6.2	0.83	[0.18-3.63]	0.784
nod_dk_pg	81	10	12.35	129	9	6.98	1.16	[0.35-3.92]	0.784
nod_skin_pg	81	11	13.58	129	12	9.3	0.87	[0.28-2.74]	0.794
nod_other_pg	81	12	14.81	129	13	10.08	0.88	[0.28-2.69]	0.798
twitch_more_	81	58	71.6	129	99	76.74	1.17	[0.30-5.55]	0.803
twitch_mr_1_	81	2	2.47	129	4	3.1	0.8	[0.07-5.78]	0.804
ft_strt_mth	81	0	0	129	0	0	1.13	[0.35-3.49]	0.809
boil_some	81	7	8.64	129	10	7.75	1.13	[0.35-3.46]	0.813
get_tw_not_w	81	21	25.93	129	32	24.81	1.09	[0.50-2.33]	0.816
fit_fell_bik	81	2	2.47	129	4	3.1	0.82	[0.07-5.96]	0.817
ed_primary	81	33	40.74	129	51	39.53	1.07	[0.57-1.98]	0.823
prklessyrly	81	1	1.23	129	2	1.55	0.76	[0.01-14.98]	0.828
faint_lst_yr	81	3	3.7	129	4	3.1	1.18	[0.17-7.20]	0.83
cyst_pst_yr	81	4	4.94	129	5	3.88	0.8	[0.07-9.91]	0.833
lrn_tw_radio	81	1	1.23	129	2	1.55	0.78	[0.01-15.30]	0.842
lrn_tw_obs	81	7	8.64	129	12	9.3	0.91	[0.29-2.64]	0.847
boil_al_al	81	1	1.23	129	2	1.55	0.8	[0.01-15.55]	0.853
any_free	81	10	12.35	129	19	14.73	1.11	[0.33-3.70]	0.855
prkneverresp	81	20	24.69	129	32	24.81	0.94	[0.46-1.93]	0.865
lat_some	81	6	7.41	129	8	6.2	1.1	[0.30-3.82]	0.865
drnktap	81	7	8.64	129	12	9.3	0.93	[0.29-2.69]	0.876
nvr_sell_pig	81	1	1.23	129	1	0.78	1.25	[0.01-104.57]	0.879
own_pig_pst_	81	12	14.81	129	18	13.95	0.92	[0.25-3.48]	0.884
skn_nod_no	81	55	67.9	129	91	70.54	0.95	[0.47-1.98]	0.89
lat_none_ob	81	11	13.58	129	16	12.4	1.06	[0.42-2.60]	0.894
epi_no	81	20	24.69	129	31	24.03	1.04	[0.51-2.11]	0.897
prk_boil	81	14	17.28	129	20	15.5	1.05	[0.44-2.43]	0.902
nod_ex_dk	81	16	19.75	129	22	17.05	0.95	[0.29-3.08]	0.916
kept_pg_any	81	37	45.68	129	60	46.51	0.97	[0.53-1.79]	0.92
hd_0	81	7	8.64	129	11	8.53	1.05	[0.33-3.13]	0.926
see_tw__med	81	9	11.11	129	14	10.85	1.05	[0.33-3.22]	0.926
dx_tw_see	81	33	40.74	129	48	37.21	1.03	[0.50-2.12]	0.928
student	81	15	18.52	129	26	20.16	0.97	[0.42-2.18]	0.93
male	81	38	46.91	129	62	48.06	0.98	[0.54-1.77]	0.937
ed_scnd_high	81	14	17.28	129	23	17.83	0.97	[0.43-2.15]	0.94
funyla	81	16	19.75	129	26	20.16	0.98	[0.45-2.06]	0.943
seizure_dk	81	3	3.7	129	5	3.88	0.95	[0.14-5.04]	0.944
sell_pig	81	18	22.22	129	34	26.36	1.06	[0.14-12.75]	0.95
othr_wrk	81	4	4.94	129	7	5.43	0.96	[0.20-4.03]	0.956
get_tw_cntct	81	3	3.7	129	5	3.88	0.96	[0.14-5.18]	0.957
sell_pglet	81	17	20.99	129	33	25.58	1.03	[0.19-7.15]	0.969

prk_fry	81	48	59.26	129	71	55.04	1.01	[0.44-2.38]	0.972
prkfrybbq	81	14	17.28	129	21	16.28	0.99	[0.42-2.27]	0.972
dx_tw_other	81	30	37.04	129	44	34.11	1.01	[0.50-2.07]	0.974
see_tw_pharm	81	10	12.35	129	16	12.4	1.01	[0.33-2.99]	0.989
lat_prt_ob	81	23	28.4	129	35	27.13	1	[0.51-1.97]	0.989
butula	81	10	12.35	129	16	12.4	0.99	[0.38-2.48]	0.99
matayos	81	22	27.16	129	35	27.13	1	[0.51-1.95]	0.996
fit_lst_yr	81	5	6.17	129	8	6.2	1	[0.25-3.62]	0.998
nod_tongue_p	81	1	1.23	129	1	0.78	1	[0.01-80.99]	1
faint_no	81	0	0	129	0	0	.	[.-.]	.
insp_alal	81	0	0	129	0	0	.	[.-.]	.
insp_al	81	0	0	129	0	0	.	[.-.]	.
amchak	81	0	0	129	0	0	.	[.-.]	.
both_pig	81	0	0	129	0	0	.	[.-.]	.
dk_pig	81	0	0	129	0	0	.	[.-.]	.
abl_brtr_cys	81	0	0	129	0	0	.	[.-.]	.
ill_after_he	81	0	0	129	0	0	.	[.-.]	.
fit_fell_wat	81	0	0	129	0	0	.	[.-.]	.
foreing_pig	81	0	0	129	0	0	.	[.-.]	.
ill_pre_hed	81	13	16.05	129	34	26.36	.	[0.00-.]	.

Appendix D: Ethical Protocol Submission to Kenya Medical Research Institute (KEMRI)

Ethical Protocol Submission for Review

Assessing the Burden of *Taenia solium* Neurocysticercosis: A Case Study of Busia District, Kenya

Submitted by

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The University of Edinburgh

November 2009

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Appendices

1.0 Title of study

Assessing the Burden of *Taenia solium* Neurocysticercosis: A Case Study of Busia District, Kenya.

2.0 The Study

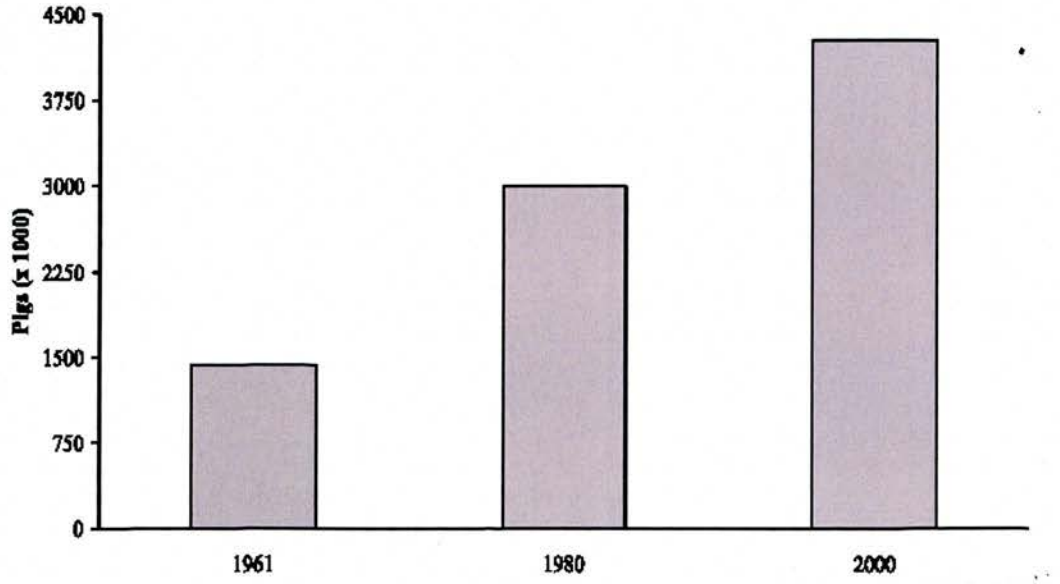
2.1 Justification for the Study

Given that there has been an increase in pig keeping in the area in recent years, and an accompanying increase in the prevalence of porcine cysticercosis it is suspected that there will be an increase in the prevalence of neurocysticercosis in Busia District. The purpose of this study is to determine that prevalence (Anyanzo 1999).

2.1.1 Emergence of pig-keeping in Western Kenya

There has been a significant increase in pig production in the Eastern and Southern Africa region (Githigia 2002); (Thuranira 2005) during the past decade, especially in rural, resource-poor, small holder communities. In Uganda and Kenya, the establishment of piggeries and increased pig production by rural farmers is encouraged by both respective governments and forms part of the plan for the modernization of agriculture under the Ministry of Agriculture in Uganda and is part of the Busia District Development Plan, also under the relevant government ministry (Government of Kenya 2002; Abila 2003). In Uganda, the local governments are supplying piglets to the rural poor communities to rear in order to promote an alternative source of income. In these communities pigs are considered low-input livestock which do not require the same level of care as cattle, for example. In addition, pigs are omnivorous; and accomplished scavengers who can grow to market size on minimal feed inputs from the farmer.

Figure 1: Trend in the total pig population in the Eastern and Southern Africa countries of Uganda, Tanzania, Kenya, Zambia, Zimbabwe and Mozambique over the past 40 years.



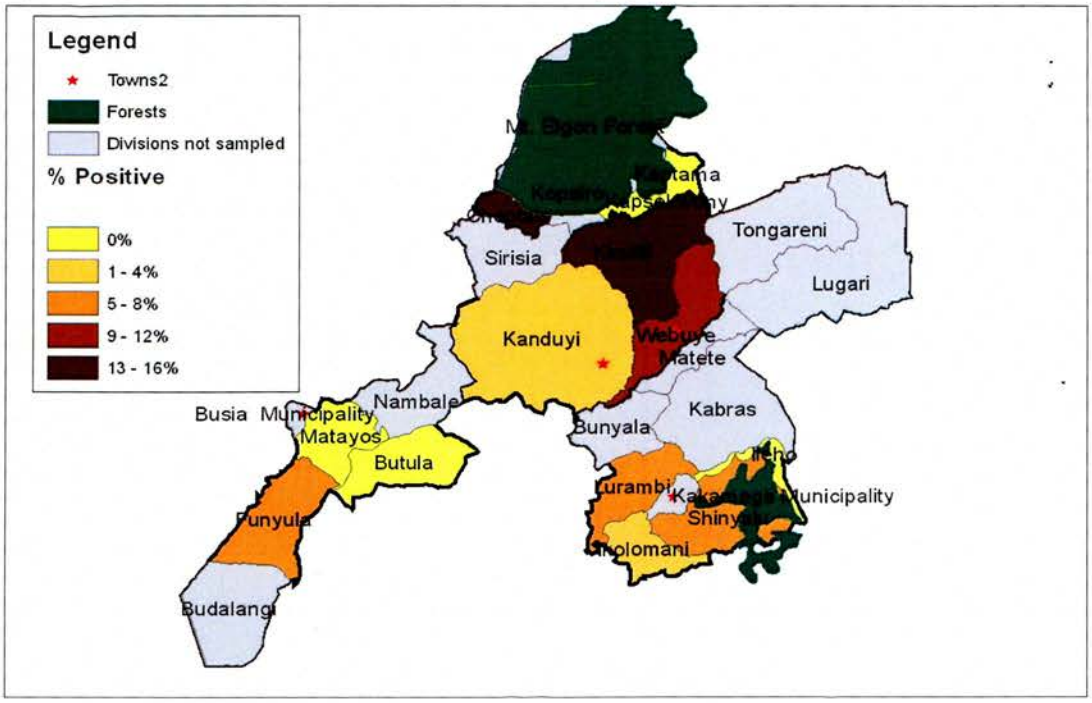
Source: (FAO 2002)

In Uganda alone, the pig population has increased from 200,000 in 1980 to almost a million in 2000 (FAO 2002). As Kenya and Uganda border each other and share many of the same cultural attributes as well as ethnicities, it would be logical to assume that there will be further increases in pig keeping likely to occur in Western Kenya.

2.1.2 Reported Increase in porcine cysticercosis

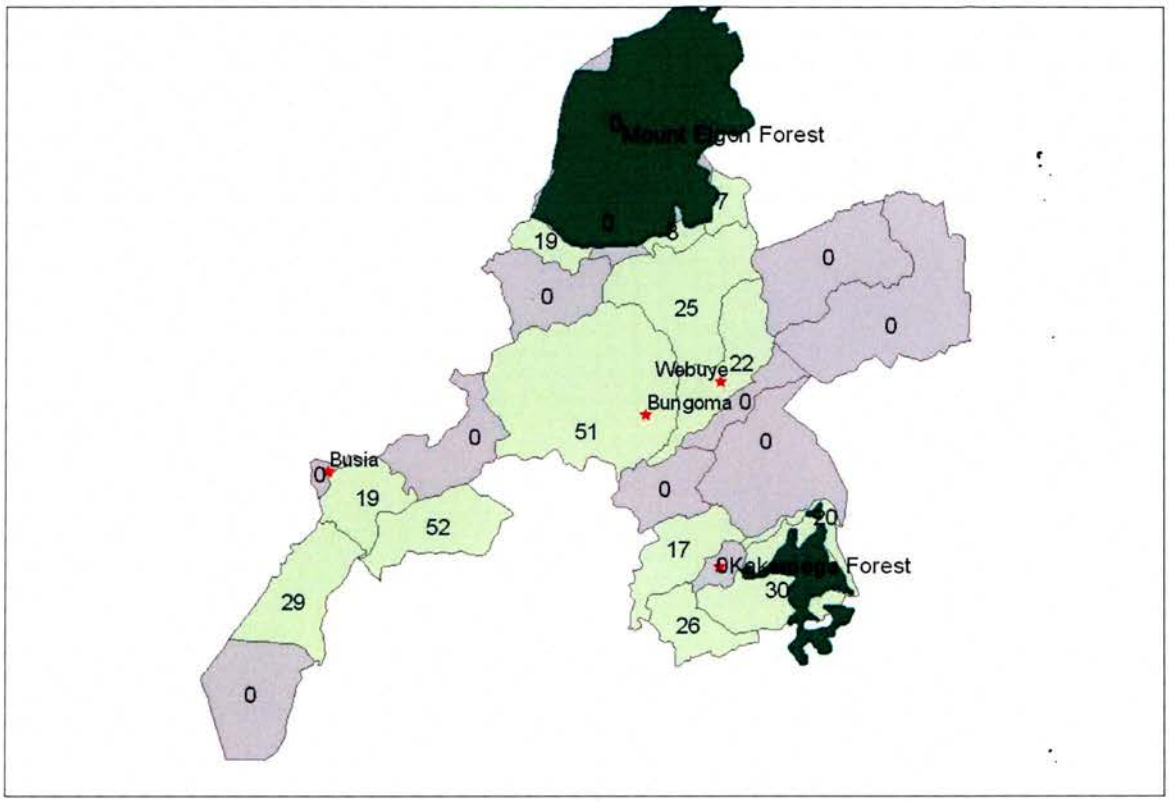
There are indications that with the increase in pig production in East Africa, porcine cysticercosis is also on the rise. In East Africa, porcine cysticercosis has been reported in Tanzania, (Nsengwa 1995), (Ngowi 2004), Kenya (Githigia 2002), (Mutua 2004) and Uganda (Anyanzo 1999), (Mafojane 2003). Ongoing work in six districts in Western Kenya has shown evidence of porcine cysticercosis in all districts (see Map 1) (Mutua 2006) with rates as high as 36% in Kimili Division using lingual examination – a method which is approximately 40% - 50% effective (Ngowi 2004). In Busia District in Western Kenya, prevalence rates for porcine cysticercosis were found ranging from 10-14% using the same diagnostic technique (Githigia 2000).

Map 1: Proportion of positive cases (prevalence) by division for the study area.



To provide information on the denominator used in calculating the division level cysticercosis prevalence rates, a map indicating the number of pigs sampled in each of the study divisions is also presented here (see Map 2 below).

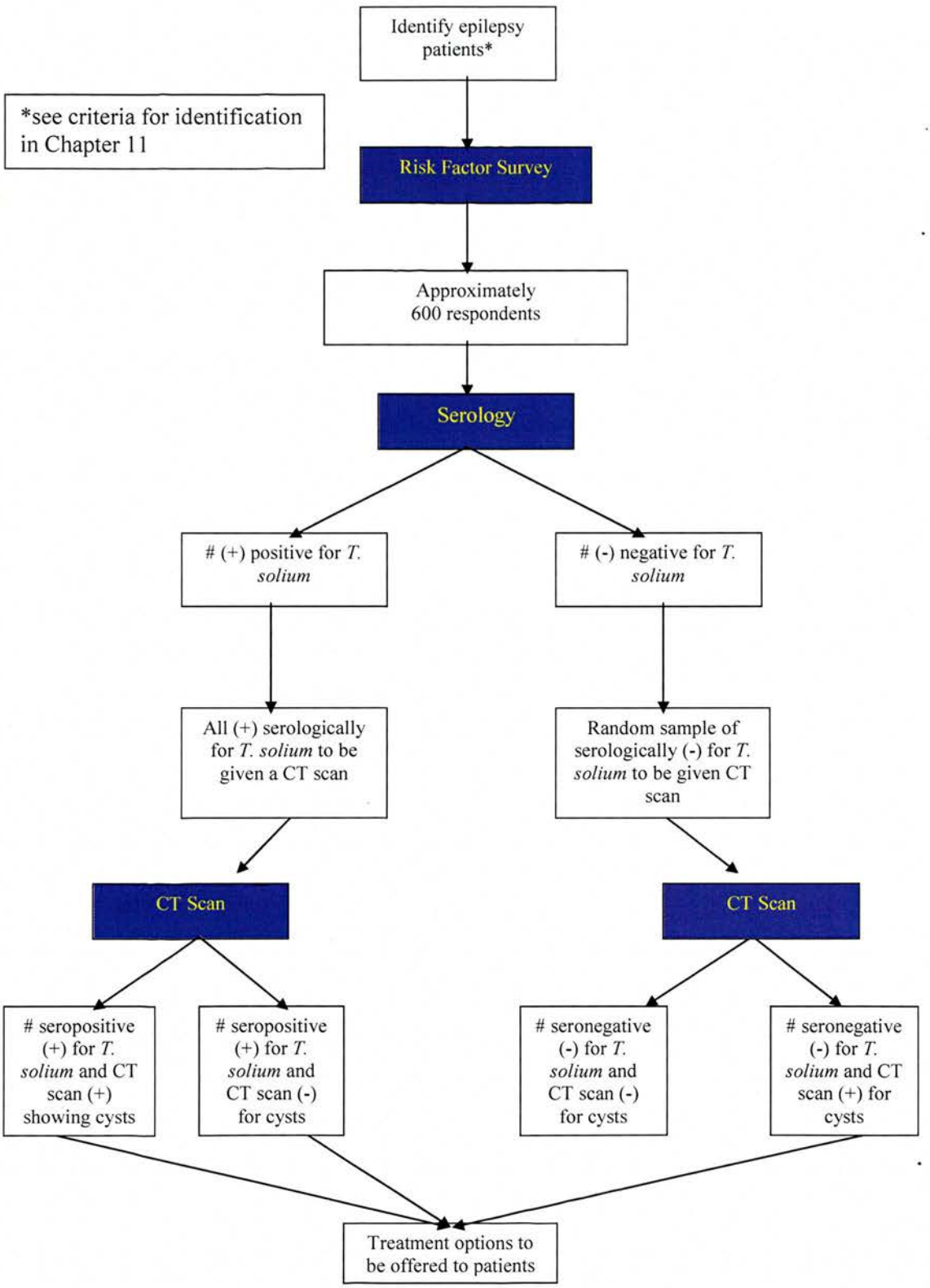
Map 2: Number of pigs sampled per division



2.2 *Objectives of the Study*

The objective of the study is to determine the prevalence of neurocysticercosis in Busia District and its impact (burden of disease) on the community. This burden will be assessed in terms of Disability-Adjusted Life Years (DALYs) for humans and livestock economic losses for pigs. To date there are almost no data available concerning the prevalence of neurocysticercosis in Kenya – only two reported cases (Phiri 2002). The following diagram indicates the process the study will follow:

Figure 2: Study Flowchart



2.3 *Hypotheses/Research Questions*

The principal hypothesis to be tested by this study is that given the increase in pig keeping in Western Kenya and the increase in porcine cysticercosis, there will be evidence of a prevalence of neurocysticercosis.

Additionally, given that the majority of the population within the study area falls below the poverty line (CBS 2003), another hypothesis will stipulate that the poor are more at risk of contracting zoonotic diseases. Once contracted, zoonotic diseases have a greater impact on the poor and are less likely to be diagnosed or treated therefore rendering the poor more vulnerable due to economic circumstances.

2.4 *Assumptions*

The assumptions associated with the study include the following:

- That the disease exists within the population
- The sample size used is sufficient to capture enough people to reach a reasonable conclusion of its presence or absence
- The diagnostics used will be sufficient and specific and sensitive enough to reach reasonable conclusions
- All measures have been taken to minimize the risks associated

To the best of our knowledge (explained in detail throughout this document) all of these assumptions are true.

2.5 *Variables*

Variables in this study that could affect the assumptions are that the prevalence that exists is significant enough to be detected and that the diagnostics used are specific and sensitive enough to do the same.

2.6 Foreseen Risks

2.6.1 Computed Tomography

As in many aspects of medicine, there are both benefits and risks associated with the use of CT (computed tomography). The main risks are those associated with abnormal test results, for a benign or incidental finding, leading to unneeded, possibly invasive, follow-up tests that may present additional risks and the increased possibility of cancer induction from X-ray radiation exposure.

The probability for absorbed x rays to induce cancer is thought to be very small for radiation doses of the magnitude that are associated with CT procedures. Such estimates of the cancer risk from x-ray exposure have a broad range of statistical uncertainty and there is some scientific controversy regarding the effects from very low doses and dose rates as discussed below. Under some rare circumstances of prolonged, high-dose exposure, x rays can cause other adverse health effects, such as skin erythema (reddening), skin tissue injury, genetic effects, and birth defects. But at the exposure levels associated with most medical imaging procedures, including CT, these other adverse effects would not occur.

Estimates of the effective dose from a diagnostic CT procedure can vary by a factor of 10 or more depending on the type of CT procedure, patient size and the CT system and its operating technique. A list of representative diagnostic procedures and associated doses are given in Table 1, adapted from a report of the European Commission (European Commission. 2000).

Table 1: Radiation Dose Comparison

Diagnostic Procedure	Typical Effective Dose (mSv)[1]	Number of Chest X rays (PA film) for Equivalent Effective Dose[2]	Time Period for Equivalent Effective Dose from Natural Background Radiation[3]
Chest x ray (PA film)	0.02	1	2.4 days
Skull x ray	0.07	4	8.5 days
Lumbar spine	1.3	65	158 days
I.V. urogram	2.5	125	304 days
Upper G.I. exam	3.0	150	1.0 year
Barium enema	7.0	350	2.3 years
CT head	2.0	100	243 days
CT abdomen	10.0	500	3.3 years

1. Effective dose in millisieverts (mSv).
2. Based on the assumption of an average "effective dose" from chest x ray (PA film) of 0.02 mSv.
3. Based on the assumption of an average "effective dose" from natural background radiation of 3 mSv per year in the United States (U.S. Food and Drug Administration. 2005).

2.6.2 Pregnant Women

All women of reproductive age will be excluded from the study. They will be asked to provide a urine sample to determine whether pregnant or not, and if found to be so, will be excluded.

2.6.3 Children

Unique Considerations for Radiation Exposure in Children

Radiation exposure is a concern in both adults and children. However, there are two unique considerations in children.

1. Children are considerably more sensitive to radiation than adults, as demonstrated in epidemiologic studies of exposed populations.
2. Children also have a longer life expectancy, resulting in a larger window of opportunity for expressing radiation damage.

As an example, compared with a 40-year old, the same radiation dose given to a neonate is several times more likely to produce a cancer over the child's lifetime. Moreover, the same exposure parameters used for a child and an adult will result in larger doses to the child. There is no need for these larger doses to children, and CT settings can be reduced significantly while maintaining diagnostic image quality. Therefore, children should not be scanned using adult CT exposure parameters. Currently, adjustments are not frequently made in the exposure parameters that determine the amount of radiation children receive from CT, resulting in a greater radiation dose than necessary.

Radiation Risks from CT in Children: A Public Health Issue

Major national and international organizations responsible for evaluating radiation risks agree there probably is no low-dose radiation "threshold" for inducing cancers, i.e., no amount of radiation should be considered absolutely safe. Recent data from the atomic bomb survivors and medically irradiated populations demonstrate small, but significant, increases in cancer risk even at the low levels of radiation that are relevant to paediatric CT scans. Doses from a single paediatric CT scan can range from about 5 mSv to 60 mSv (see Table 2). Among children who have undergone CT scans, approximately one-third have had at least three scans. Multiple scans present a particular concern. For example, three scans would be expected to triple the cancer risk of a single scan.

Although the benefits of properly performed CT examinations almost always outweigh the risks for an individual child, unnecessary exposure is associated with unnecessary risk. Minimizing radiation exposure from paediatric CT, whenever possible, will reduce the projected number of CT-related cancer deaths.

Table 2.: Radiation Doses Paediatric CT scan

EXAMINATION TYPE	RELEVANT ORGAN	APPROXIMATE EQUIVALENT DOSE TO RELEVANT ORGAN (mSv)
Paediatric Head CT Scan Unadjusted Settings* (200 mAs, neonate)	Brain	60
Paediatric Head CT Scan Adjusted Settings* (100 mAs, neonate)	Brain	30
Pediatric Abdominal CT Scan Unadjusted Settings (200 mAs, neonate)	Stomach	25
Pediatric Abdominal CT Scan Adjusted Settings (50 mAs, neonate)	Stomach	6
Chest X-ray (PA/lateral)	Lung	0.01 / 0.15
Screening Mammogram	Breast	3

* *"Unadjusted" refers to using the same settings as for adults. "Adjusted" refers to settings adjusted for body weight.*

Source: (National Cancer Institute. 2006)

Immediate Measures to Minimize CT Radiation Exposure in Children

Physicians, other paediatric health care providers, CT technologists, CT manufacturers and various medical and governmental organizations share the responsibility to minimize CT radiation doses to children. Several immediate steps can be taken to reduce the amount of radiation that children receive from CT examinations:

- Perform only necessary CT examinations. Communication between paediatric health care providers and radiologists can determine the need for CT and the technique to be used. Although there are standard indications for CT in children, radiologists should review reasons prior to every paediatric scan and be available for consultation when indications are uncertain. Consider other modalities such as ultrasound or magnetic resonance imaging, which do not use ionizing radiation.

- Adjust exposure parameters for paediatric CT based on:
  - Child size: guidelines based on individual size / weight parameters should be used.
  - Region scanned: the region of the body scanned should be limited to the smallest necessary area.
  - Organ systems scanned: lower mA settings should be considered for skeletal and lung imaging.
  - Scan resolution: the highest quality images (i.e., those that require the most radiation) are not always required to make diagnoses. In many cases, lower-resolution scans are diagnostic.

- Minimize the CT examinations that use multiple scans obtained during different phases of contrast enhancement (multiphase examinations). These multiphase examinations are rarely necessary, especially in body (chest and abdomen) imaging, and result in a considerable increase in dose (National Cancer Institute. 2006)

While acknowledging the previously stated risks with regard to paediatric CT scans, it is unlikely that the exposure to radiation from this one scan will do significant long term damage and that the benefits in terms of the child's potential to be treated and relieved of epileptic symptoms, clearly outweigh the risks.

3.0 Information on previously published research on the topic

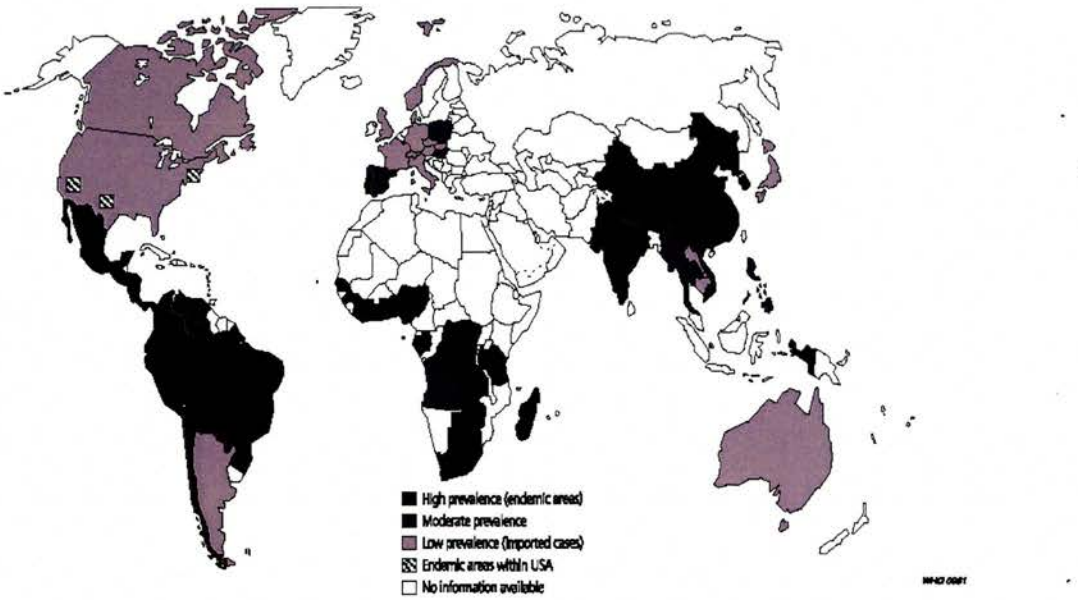
3.1 Worldwide status of cysticercosis

Cysticercosis was originally a ubiquitous disease occurring wherever pigs and humans existed in association, and is probably a disease of great antiquity – Aristotle gives a clear description of the condition as it occurs in pigs (Cook 2003). Although cysticercosis has been known for many years, its relationship with the adult tapeworm appears not to have been clear until it was demonstrated by Kuchenmaister in 1855 when he fed condemned prisoners with cysticercosis-infected pork and recovered young tapeworms at the autopsies (Henneberg 1912). There is, however, some evidence to suggest that as early as the 6th century there may have been some

knowledge about it as illustrated by the example of Anthinus who reported to Theodoric, King of the Franks (511-534), that "tapeworms were excreted after the consumption of raw fatty pork" (Gach 1926). Even if this connection had been established, there is no evidence that a link was made between the tapeworm transmitted and its effects on humans.

The disease has been reported in West African countries such as Benin, Burkina-Faso, Ghana, Ivory Coast, Senegal and Togo and is expected to be present in most pig-raising regions of other West African countries. In the Nsukka area of Enugu State in Nigeria, over 20% of 2358 trade pigs examined were found infected with porcine cysticercosis and the overall prevalence of taeniid ova in the 1525 human stool samples analysed was 8.6% with most of the cases (78.6%) occurring adults aged >30 years (Onah 1995). Although economic losses are associated with the decreased value of a pig infected with cysticercosis (Zoli 2003), it has to be noted that in some villages in West Cameroon, pork infected with cysticerci is considered to have a better flavour than healthy meat. Therefore, pork harbouring cysticerci is sometimes sold at a higher price than uninfected meat (Zoli 1992).

Map 3: Areas where cysticercosis is endemic



Countries in black represent countries where cysticercosis is endemic; countries in grey represent those where cases have been reported, (Roman 2000).

3.1.1 History of cysticercosis in East Africa

In East Africa, the disease has been reported in Tanzania, Kenya and Uganda. In Tanzania, a retrospective study of slaughter records in Mbulu district from 1985-1989 indicates a prevalence increasing from 0.4 to 4.9% during that time (Nsengwa 1995). In a more recent study of the risk factors associated with cysticercosis in Mbulu District, pigs raised in a total of 436 households were examined in the 21 selected villages in the district. Out of the 770 pigs examined, 134 had visible *T. solium* larvae, giving an overall prevalence of 17.4% (95% CI = 12.5, 22.3). The prevalence in the 21 villages ranged from 3.2 – 46.7%, showing a large variation in villages (Ngowi 2004).

In Kenya, the disease has not been considered endemic for several decades but recent surveys indicate that *T. solium* is emerging as a problem in small-holder pig keeping communities of south-western Kenya where ante-mortem lingual examinations in different locations in the area have indicated at least 10-14% of the pigs surveyed to be infected (Githigia 2002). According to the Annual Reports of the Ministry of Health (MOH), the prevalence of human taeniosis in the rural areas is estimated to be 2% (Mafojane 2003) but could be as high as 4-10% in Busia District as hospital records suggest (Busia District Hospital 2001).

In Uganda, there is not enough data to quantify the prevalence of cysticercosis, but according to a post-mortem survey conducted in 1999 in northern Uganda, 34-45% of the pigs slaughtered were infected. Given the increases in pig-keeping in Uganda over the past 20 years and the prevalence of porcine cysticercosis in neighbouring countries, it is likely that there will soon be a public health problem (Mafojane 2003).

3.2 Neurocysticercosis (NCC)

3.2.1 General Background to NCC

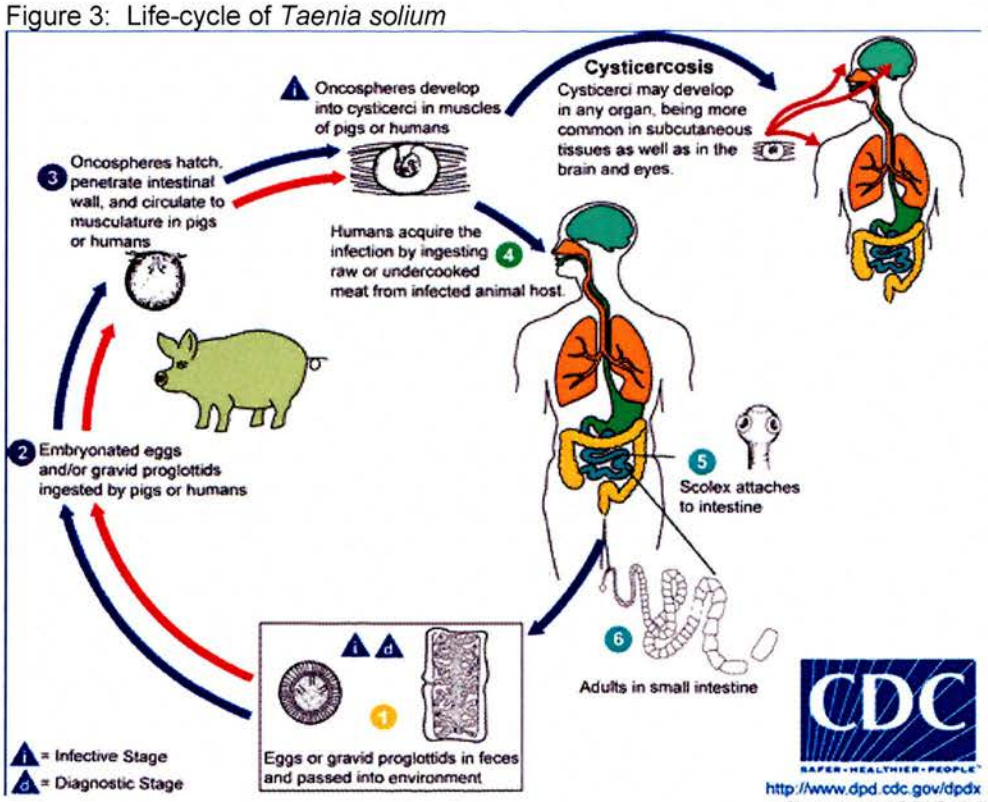
Neurocysticercosis (NCC) is a parasitic infection that can affect the nervous system caused by *Taenia solium,* which is common, but preventable. In both humans and pigs, a therapeutic does of drugs is the most common form of treatment but often

detection is under-reported (Tsang 1995) and better surveillance and monitoring is necessary to control the infection. Infection with *T. solium* is widely prevalent in human and pig hosts in many developing countries of Latin American, Africa, and Asia (Sarti 2003).

Humans are the only natural definitive host while pigs are the intermediate host. Man may become an intermediate host from ingestion of the eggs of the adult tapeworm, resulting in a condition known as human cysticercosis. The cysticerci of *T. solium* may lodge in the brain causing cerebral cysticercosis (neurocysticercosis), resulting in headaches, epileptic seizures, blindness and mental disturbances (White 2000). In addition, the cysticerci may cause other neurological afflictions such as meningitis, encephalitis, ventricular disease, spinal disease and ocular cysticercosis, although the most common manifestation still remains epilepsy (Cook 2003).

The *T. solium* taeniosis/cysticercosis complex is associated with poor sanitation and hygiene, poor methods of pig-husbandry and lack of proper meat inspection and disease control methods. Ingestion of larvae (cysticerci) in raw or under-cooked pork meat results in the human tapeworm infection or taeniosis (Phiri 2003).

Figure 3: Life-cycle of *Taenia solium*



Source: (CDC 2004)

Figure 4: Description of the life cycle of *Taenia saginata* and *Taenia solium*

Humans are the only definitive hosts for *Taenia saginata* and *Taenia solium*. Eggs or gravid proglottids are passed with feces 1; the eggs can survive for days to months in the environment. Cattle (*T. saginata*) and pigs (*T. solium*) become infected by ingesting vegetation contaminated with eggs or gravid proglottids 2. In the animal's intestine, the oncospheres hatch 3, invade the intestinal wall, and migrate to the striated muscles, where they develop into cysticerci. A cysticercus can survive for several years in the animal. Humans become infected by ingesting raw or undercooked infected meat 4. In the human intestine, the cysticercus develops over 2 months into an adult tapeworm, which can survive for years. The adult tapeworms attach to the small intestine by their scolex 5 and reside in the small intestine 6. Length of adult worms is usually 5 m or less for *T. saginata* (however it may reach up to 25 m) and 2 to 7 m for *T. solium*. The adults produce proglottids which mature, become gravid, detach from the tapeworm, and migrate to the anus or are passed in the stool (approximately 6 per day). *T. saginata* adults usually have 1,000 to 2,000 proglottids, while *T. solium* adults have an average of 1,000 proglottids. The eggs contained in the gravid proglottids are released after the proglottids are passed with the feces. *T. saginata* may produce up to 100,000 and *T. solium* may produce 50,000 eggs per proglottid respectively.

Source: (CDC 2004)

T. solium has a complex two-host life cycle. Human beings are the only definitive host and harbour the adult tapeworm (taeniosis), whereas both people and pigs can act as intermediate hosts and harbour the larvae or cysticerci (Garcia 2003). It is possible, however, for someone who has never eaten pork to contract neurocysticercosis by a process of "auto-infection". This would mean that the individual could become infected by ingesting a tapeworm segment excreted by themselves or another human which could be found contaminating other types of food and not pork. The segment would then develop into cysticerci in the muscles of the human and then move to the central nervous system (CNS) causing neurocysticercosis (Schantz 1992).

Approximately 2.5 million people worldwide carry adult *T. solium* (Burneo 2001). The prevalence of epilepsy in industrialized countries is about 3-9 per 1000 population. The prevalence in developing countries, data which is based largely on community surveys of rural populations, is as high as 49 per 1000 population in Liberia and 57 per 1000 in Panama (Senanayake 1993).

According to the International League Against Epilepsy (ILAE), cysticercosis is probably the most common cause of acquired epilepsy in the developing world, where prevalence rates of active epilepsy are twice those of developed countries (ILAE 2006). Cysticercosis is important in smallholder farming communities because it exacts a price by:

- Causing infections in humans affecting their physical and psychological health, social life and productivity;
- Seriously reducing farmers' household incomes due to potential condemnation of carcasses.

3.3 Epilepsy

3.3.1 General Background

The word "epilepsy" derives from the Greek verb "epilambanein", meaning "to be seized, to be overwhelmed by surprise" and captures well the sudden, usually unpredictable and intrusive nature of most seizures. Neurologists define epilepsy as:

"a condition in which individuals experience paroxysmal changes in behaviour caused by abnormalities in the electrical activity of the brain" (Asbury 1992). In other words, epilepsy is the name given to a group of functional disorders of the brain that are characterized by repetitive seizures. Seizures involve abnormal, excessive electric discharges of groups or assemblies of nerve cells (neurones) in the brain (WHO 2004).

Epilepsy is the most common serious neurological disorder and is one of the most prevalent non-communicable disease in the world (Scott 2001). The WHO Neurosciences Research Protocol for studying the prevalence of neurological disorders in developing countries defines epilepsy as two or more afebrile seizures unrelated to acute metabolic disorders or to withdrawal of drugs or alcohol (Senanayake 1993). According to the WHO "Out of the Shadows" – Global Campaign Against Epilepsy, in all areas of the world, no less than three out of every thousand people – and in several places over 40 per thousand (4%) are affected.

3.3.2 Prevalence in Sub Saharan Africa

The prevalence of epilepsy for sub-Saharan Africa varies considerably, with smaller studies showing in general a higher prevalence and larger studies showing prevalence nearer that in the developed world. The median prevalence found using door-to-door studies is 15 per 1000 people (Preux 2005). The table below illustrates varying rates of prevalence using door-to-door studies in sub-Saharan Africa.

Table 3: Prevalence of epilepsy in sub-Saharan Africa

Country	Reference	Year	N	Prevalence %	95 % CI
Benin (Agbogbomé)	(Gbenou 1995)	1995	530	24.5	10.9-38.1
Benin (Savalou)	(Avodé 1996)	1996	1,443	15.2	8.7-21.7
Benin (Zinvié)	(DeBrock 2000)	2000	3,134	33.5	22.3-44.3
Burkina Faso	(Debouverie 1993)	1993	16,627	10.6	9.1-12.2
Cameroon	(Nkwi 1989)	1989	500	70.0	46.3-93.6
Cameroon (Bilomo)	(Dongmo 2000)	2000	1,900	58.4	46.9-69.1
Ethiopia (Butajira)	(Tekle-Haimanot 1990)	1990	60,820	5.2	4.6-5.8
Ivory Coast	(Kouassi 1988)	1988	1,176	7.6	2.5-12.7
Ivory Coast	(Kouadjo 1990)	1990	309	74.4	43.0-104.9
Ivory Coast (M'Brou)	(Kaudjhuis 1995)	1995	920	59.0	43.0-75.0
Kenya (Nakuru)	(Kaamugisha 1988)	1988	2,960	18.2	13.2-23.2
Liberia	(Goudsmit 1983)	1983	4,436	28.0	23.0-33.0
Madagascar	(Andriantseheno 2004)	2001	925	20.8	11.3-30.3
Mali	(Farnarier 2000)	2000	5,243	15.6	12.2-19.0
Mali (Bamako)	(Traoré 2000)	2000	4,074	11.3	8.0-14.6
Nigeria	(Longe 1989)	1989	2,925	6.2	3.3-9.1
Nigeria (Aiyété)	(Osuntokun 1982)	1982	903	37.0	24.2-49.8
Nigeria (Igbo-Ora)	(Osuntokun 1987)	1987	18,954	5.3	4.2-6.4
Senegal	(Ndiaye 1986)	1986	7,682	8.3	6.2-10.4
Senegal	(Diop 1996)	1996	2,803	21.0	15.5-26.5
Tanzania	(Rwiza 1992)	1992	18,183	10.2	8.7-11.7
Togo (Akebou)	(Grunitzky 1996)	1996	4,182	13.1	9.6-16.6
Togo (Kloto)	(Grunitzky 1991)	1991	19,241	12.3	10.7-13.9
Togo (Kozah)	(Dumas 1989)	1989	5,264	16.7	13.1-20.3
Togo (Tone)	(Balagou 2000)	2000	9,143	18.6	15.7-21.5
Uganda	(Kaiser 1996)	1996	4,743	13.0	9.7-16.3
Zambia (Chikankata)	(Birbeck 2004)	2004	55,000	12.5	11.5-13.5

Source: (Preux 2005)

It is estimated that 10% of the burden of brain and mental disorders in the world is caused by epilepsy, calculated in disability-adjusted life years (DALYs), which is very significant. This calculation includes premature deaths and the loss of healthy life due to disability (World Bank 1993). Every year, among every 100,000 persons, there will be 40-70 new cases (WHO 2003). Of the 50 million people in the world who have epilepsy, approximately 35 million have no access to appropriate treatment. This is in most cases because services are unaffordable or non-existent and in some cases because epilepsy is not viewed as a medical problem or as a treatable brain disorder (WHO 2001b).

3.3.3 Burden of Disease Estimates for Epilepsy

The burden of disease (BOD) estimates for epilepsy include epilepsy and status epilepticus. (Mathers 2003) estimate the DALYs for epilepsy as 6,223,000, with slightly higher rates for males (3,301,000) than for females (2,922,000). Many risk factors for epilepsy are linked with a lower level of economic development; thus, the burden is highest in South Asia followed by Sub-Saharan Africa (Table 4). A notable observation is the reportedly low burden in the Middle East and North Africa, despite parts of that region being relatively underdeveloped. Epilepsy imposes a large economic burden on patients and their families. It also imposes a hidden burden associated with stigmatization and discrimination against patients and even their families in the community, workplace, school, and home. Social isolation, emotional distress, dependence on family, poor employment opportunities, and personal injury add to the suffering of people with epilepsy (Jamison 2006).

Table 4: Disability-Adjusted Life Years by Cause and Region, 2001 (x1000)

	Global total									
Condition	Mixed sexes	Males	Females	East Asia & Pacific	Europe & Central Asia	Latin America & the Caribbean	Middle East & North Africa	South Asia	Sub-Saharan Africa Countries	High-income Countries
AD and other Dementias	17,108	6,092	11,016	4,110	1,612	1,215	292	1,955	450	7,468
Epilepsy	6,223	3,301	2,922	1,303	354	737	248	1,741	1,373	464
PD	2,325	1,124	1,202	435	228	90	81	303	100	1,086
Cerebrovascular Disease	72,024	35,482	36,542	25,832	12,616	3,936	1,948	13,184	5,125	9,354

Source: (Mathers 2003)

More than 80% of patients with epilepsy live in developing countries and most of these live in sub-Saharan Africa (WHO 2001b). Data on the incidence of and prognosis for epilepsy in sub-Saharan Africa are scarce but prevalence data show that epilepsy is two to three times more common than in industrialized countries in non-tropical areas (Preux 2005).

3.3.4 Definitions for Epilepsy Diagnosis

Epileptic seizure. A clinical manifestation presumed to result from an abnormal and excessive discharge of a set of neurons in the brain. The clinical manifestation consists of sudden and transitory abnormal phenomena which may include alterations of consciousness, motor, sensory, autonomic, or psychic events, perceived by the patient or an observer.

Epilepsy. A condition characterized by recurrent (two or more) epileptic seizures, unprovoked by any immediate identified cause. Multiple seizures occurring in a 24-h period are considered a single event. An episode of status epilepticus is considered a single event. Individuals who have had only febrile seizures or only neonatal seizures as herein defined are excluded from this category.

Status epilepticus. A single epileptic seizure of >30-min duration or a series of epileptic seizures during which function is not regained between ictal events in a >30-min period.

"Active" epilepsy. A prevalent case of active epilepsy is defined as a person with epilepsy who has had at least one epileptic seizure in the previous 5years, regardless of antiepileptic drug (AED) treatment. A case under treatment is someone with the correct diagnosis of epilepsy receiving (or having received) AEDs on prevalence day.

Epilepsy in remission with treatment. **A** prevalent case of epilepsy with no seizures for ≥ **5** years and receiving AED at the time of ascertainment.

Epilepsy in remission without treatment. A prevalent case of epilepsy with no seizures for ≥5 years and not receiving AED at the time of ascertainment.

Single or isolated seizure. One or more epilepticseizures occurring in a 24-h period.

Febrile seizure. An epileptic seizure as herein defined, occurring in childhood after age 1 month, associated with a febrile illness not caused by an infection of the CNS, without previous neonatal seizures or a previous unprovoked seizure, and not meeting criteria for other acute symptomatic seizures.

Neonatal seizure. An epileptic seizure as herein defined occurring in the first **4** weeks of life.

Febrile seizure with neonatal seizure. One or moreneonatal seizures in a child who has also experienced one or more febrile seizures as herein defined.

Nonepileptic events. Clinical manifestations presumed to be unrelated to an abnormal and excessive discharge of a set of neurons of the brain, including:

- disturbances in brain function (vertigo or dizziness, syncope, sleep and movement disorders, transient global amnesia, migraine, enuresis), and pseudoseizures (nonepileptic sudden behavioral episodes presumed to be of psychogenic origin; these may coexist with true epileptic seizures).

All definitions have been taken from the Commission on Classification and Terminology of the International League Against Epilepsy (ILAE 1989).

4.0 Previous Submissions

There have been no previously submitted protocols.

5.0 Description of Study Site

5.1 Background to Busia District

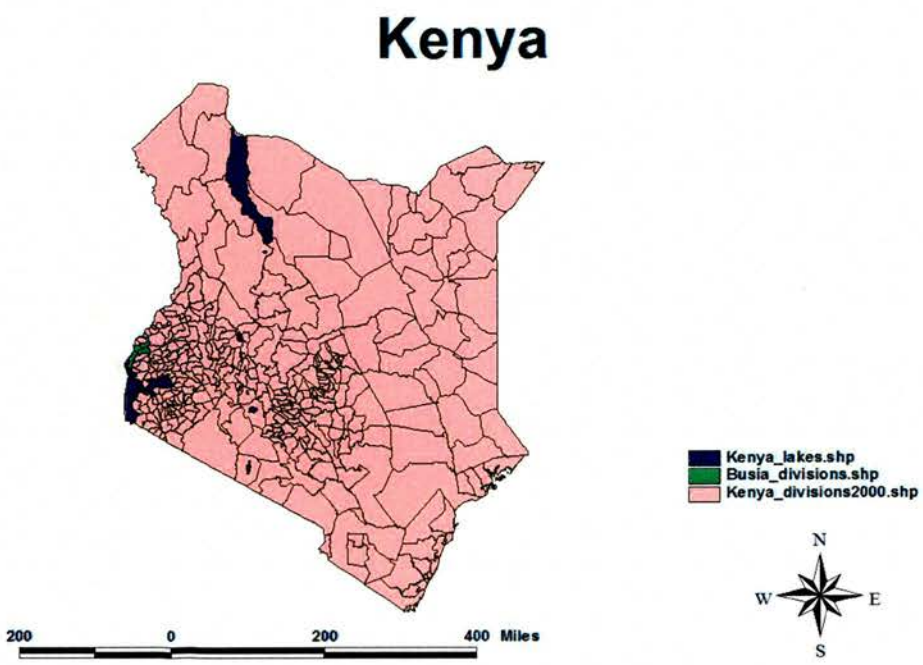
Busia District is located in Western Kenya, bordering Uganda on the west side, Teso District to the north, Siaya and Mumias Districts to the east and Lake Victoria to the south. Busia District falls within the Lake Victoria Basin.

The altitude varies from 1 130 m above sea level on the shores of Lake Victoria to 1 375 m above sea level in the central part. The northern divisions (Butula and Nambale) occupy a plain characterised by low flat divides. These are often capped by laterites and shallow incised swampy systems. The peneplain has fertile soils suitable for growing maize, Robusta coffee and sugar cane.

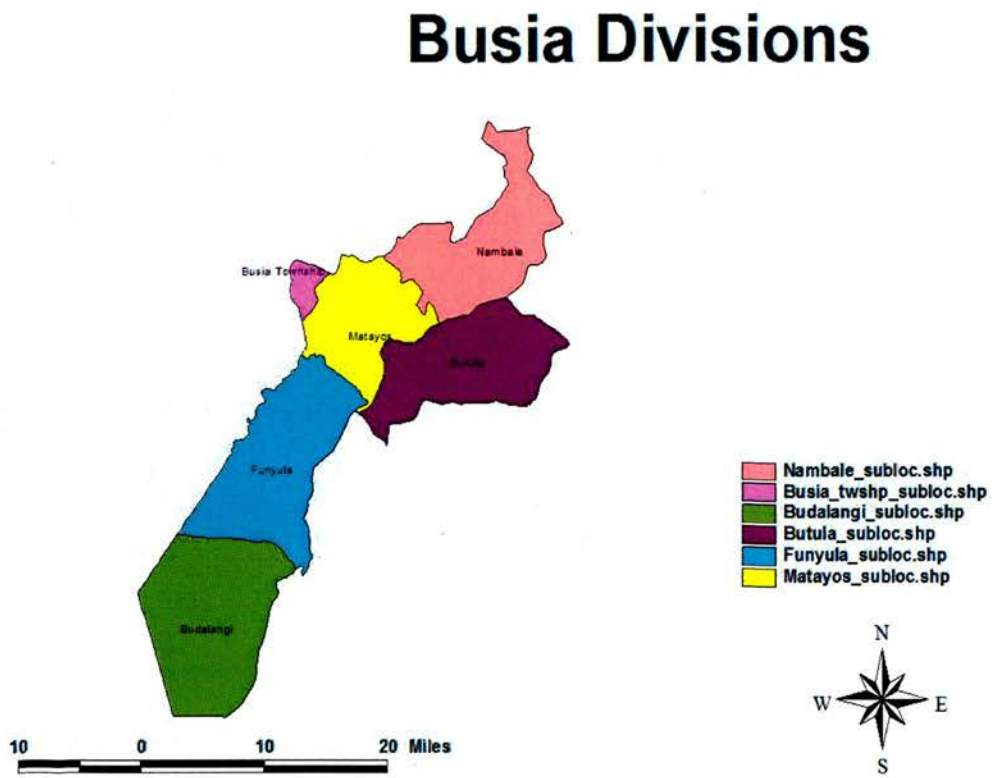
The southern part, which covers parts of Matayos Division, Funyula Division and the northern part of Budalang'i Division is covered by a range of hills comprising the Samia Hills, which run from northeast to southwest, culminating at Port Victoria. The Yala Swamp is found in the extreme south of the district, an area which forms a colony of papyrus growth broken by irregular water channels and occasional small lakes with grassy islands (CBS 2000). Busia has long been a tsetse endemic area (Hide 1999) and in recent years, has seen a change in livestock keeping patterns with more market oriented livestock such as pigs on the increase. The human population of Busia District is currently reported to be in the region of 405,000 with the most populous division being Butula Division.

The district is divided into six divisions: Funyula, Butula, Nambale, Matayos, Budalang'i and Busia Municipal (see Maps 4 & 5).

Map 4: Kenya with Busia District



Map 5: Busia District with Divisions



Recent surveys indicate that although not considered endemic in Kenya for several decades, with the emergence of small holder pig keeping communities in south-western Kenya (Busia, Kakamega), *T. solium* prevalence is increasing (Phiri 2003). In addition, Tanzania is considered a cysticercosis endemic country (Roman 2000) and the area where the disease is particularly prolific is located relatively close to the Kenyan border (Ngowi 2004). The potential for cases of porcine cysticercosis to increase is being attributed to poor sanitation and the relaxation of enforcement of regulations concerning the management of pigs, in particular allowing free range pig keeping (Githigia 2002).

Table 5: Human Population Densities by Division

Division	1999				2002			2008	
	Area (km^2)	Population	Density		Population	Density		Population	Density
Budalang'i	306.5	53 356	174		58 363	190		69 836	228
Butula	245.2	95 489	389		104 450	426		124 970	510
Funyula	281.2	73 875	263		80 808	287		96 687	344
Matayos	173.7	55 186	318		60 365	348		72 227	416
Nambale	232.5	67 544	291		73 883	318		88 401	380
Township	22.2	25 158	1 133		27 519	1 240		32 926	1 483
Totals	**1 261.3**	**370 608**	**294**		**405 389**	**321**		**485 047**	**385**

Source: (CBS 1999) and District Statistics Office, Busia, 2001.

6.0 Name and address of the sponsor;

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7.0 Investigators

(Curriculum Vitaes attached as Appendix I)

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8.0 Study Type

The study to be conducted will be a case-control study. The sample population will be those who have previously been diagnosed with epilepsy. The diagnosis of epilepsy itself will not be part of the study as it will have been carried out previously by qualified Kenyan medical staff. The cases will be those who have tested positive serologically for exposure to *T. solium* and the control group will be those subjects who have tested negatively serologically for exposure to *T. solium*.

Table 6: Contingency table for Case-Control Study

	Diseased subjects (+ *T. solium*)	**Non-diseased subjects (- *T. solium*)**	**Total**
Hypothesized risk factor present (e.g., Latrine not enclosed)	***A***	***B***	***a + b***
Hypothesized risk factor not present (e.g.,Latrine enclosed)	***C***	***D***	***c + d***
Total	***a + c***	***b + d***	***a + b + c +d = n***

The contingency table for a case-control study assumes the following (***a+c***) and ***(b+d)***, are pre-determined.

Source: (Thrusfield 1995)

All those who have tested positively will be given a CT scan at the Aga Khan Hospital in Kisumu to determine the presence of cysts in the brain along with a random sample of those who have tested negatively. The scans will be analysed by a qualified Radiologist, Dr Reddi Rao, and the results given to the study subjects and the investigators. All results will be kept confidential by the invested parties.

8.1 CT Scan Protocol

The protocol for the CT scan will be as follows:

- 5mm cuts in the posterior fossa
- 10mm cuts in the supratentonlotium
- Pre and post contest scans (contrast required)
- Coronal sections if required

8.2 Risk Factor Investigation

The risk factors deemed to be present or absent have been determined by administration of a questionnaire to the same sample population of epileptic patients (see Appendix 2). These risk factors include:

- Gender
- Educational levels
- Pork Consumption
- Presence and use of a latrine (enclosed totally, partially, not at all)
- Pig husbandry practices (tethered at certain times, total free range)
- Meat slaughter and inspection practices
- Familiarity with both porcine and human cysticercosis
- Familiarity with *Taenia solium*
- Diagnosis of epilepsy
- Symptoms of epilepsy

A follow-up set of questions designed to firstly, validate some of the answers to the first questionnaire and, secondly, determine whether even if they are not directly exposed to pigs themselves, their physical proximity to pigs in the area (Appendix IX). At this time, an Asset Index (Appendix X) will also be discussed with the respondents to establish their economic status.

9.0 Number of Participants – Statistically Determined

As indicated in Table 1, prevalence rates for epilepsy in sub-Saharan Africa range from 5.2% to 74.4% using different sample sizes in door-to-door surveys. The prevalence rate for Kenya set out in this table for epilepsy is 18%. Infections are the

cause of epilepsy in up to 26% of the patients included in Table 1 (Preux 2005). While recent reviews have noted the absence of well-conducted studies that can establish the importance of neurocysticercosis in epilepsy (Pal 2000), it is considered the major cause of acquired epilepsy in Latin America (Garcia, Gilman *et al.* 1993), and may also be so in sub-Saharan Africa (Dumas 1989), (Nsengiyumva 2003). In Nsengiyumva's study carried out in Kiremba, Burundi, of the cases (people with epilepsy), 59.6% were positive for ELISA cysticercosis serology and of the controls (those without diagnosed epilepsy), 31.5% were positive for cysticercosis. The sample size in this case was 324 cases and 648 controls. In a similar study conducted by Newell in Bururi, Burundi, 4.9% of the 103 epileptic cases and 4.2% of the 72 controls showed exposure to *T. solium* in Ag ELISA (Newell, Vyungimana et al. 1997). The following table illustrates some of the risk factors for acquiring epilepsy in sub-Saharan Africa.

Table 7: Case-control studies of risk factors for epilepsy in sub-Saharan Africa

Country	Reference	Year	Patients with Epilepsy	Controls	OR FS	OR FH	OR HI	OR PC	OR cysticercosis
Burundi	(Newell, Vyungimana et al. 1997)	1997	103	72					4.6
Burundi	(Newell, Vyungimana et al. 1997)	1997	110	82					
Burundi	(Nsengiyumva 2003)	2003	324	648		3.3		1.9	4.1
Cameroon	(Dongmo 2004)	1998	93	81					NS
CAR	(Druet-Cabanac 1999)	1999	187	374					
Mali	(Farnarier 2000)	2000	70	140					
Nigeria	(Ogunniyi 1987), (Bademosi 1989)	1989	155	155	11.0		13.0		
Tanzania	(Matuja 2001)	2001	174	174	2.9	3.3		4.5	

OR=odds ratio; FS=febrile seizures; FH=family history; HI=head injury;
PC=perinatal complications; NS=not significant; CAR=Central African Republic

Source: (Preux 2005)

In the proposed study approximately 600 epileptic patients will be tested for exposure to *T. solium* using Ag ELISA. Those who test positively will be given a CT scan as well as a doubled random number of those who test negatively for exposure to *T. solium*.

A sample size of around 600 patients is sufficient to detect a prevalence of 15% (Kaamugisha 1988); (Kaiser 1996); (Rwiza 1992)), (estimated from the literature) with an error of 5% at 95% confidence, based on an estimated population in Busia District of 400,000 (CBS 2000).

From this sample of epileptics, a case control study will be designed to determine risk factors for NCC induced epilepsy among epileptics in Busia. The exact nature of the study design for this section of the study will depend in part on the results of the serology; either a matched or un-matched case control design will be used, requiring 205 or 294 cases respectively.

10.0 Exclusion or Inclusion of Subjects

The only participants who will be excluded from the study are women who test positively for pregnancy or express that they intend to becom pregnant during the course of the study.

11.0 Process of Recruitment

A pilot phase of the study has already been carried out which involved identifying study subjects based on their attendance at a facility that treats epileptics. This part of the study is intended as the pilot phase on the feasibility of the overall study. The Chief Medical Officer, Dr James Mukabi was informed of the study and provided a letter of introduction to facilitate collecting information from the records of health facilities (Appendix III). In this pilot study, approximately 400 patients were identified at six facilities around Busia District. These facilities were St Catherine's Special School in Butula, St Martin's Special School in Kisoko, Nangina Special School in Funyula, Port Victoria Sub-district Hospital in Budalang'i, and Busia District Hospital, Busia.

The diagnosis for epilepsy was therefore carried out by a qualified physician and the patients were on a regime of anti-epileptic drugs (AEDs). The intention of the study is to recruit at least 600 patients using this method to have an adequate sample size.

It must be noted, however, that as the patients are attending a health facility, they must have the ability to pay for these services. This method of recruitment clearly precludes those who have epilepsy but lack the means to seek treatment. As poverty is a risk factor associated with the transmission of *T. solium* (Garcia 2003; Ngowi 2004), it may be assumed that this excluded population may in fact be one most at risk of infestation. It will then be necessary to recruit subjects who are perhaps known by other epileptics but not by the health officers or who are known by their communities. If these patients are on drug therapy prescribed for epileptics they will be classified as diagnosed epileptics.

One of the objectives of the study is to administer a questionnaire (Appendix II) to determine risk factors for the transmission of *T. solium*. This questionnaire will include questions regarding symptoms of epilepsy to assess whether a subject has epilepsy but has not been formally diagnosed by a qualified physician but in fact has epilepsy.

11.1 Steps taken to assure confidentiality during recruitment

As many of the patients will be identified by fellow epileptics as having similar problems as theirs, it will be impossible for the identity of epileptic patients to be suppressed entirely. Care will be taken, however, to confine the questionnaire to the epileptic patient and/or their guardians and to preserve confidentiality when interviewing patients.

12. Samples of the Standardized Case Report Forms

Appendix IV

13. Liability for research-related disability or death

The research involved does not carry more than a minimal risk of physical injury.

14. Medical Care for Subjects

Prior to the collection of sera from the epilepsy patients, a training session will be held in Busia District for clinical officers, psychiatric officers and others who are responsible for care of epileptic patients and *Taenia solium* related conditions. The training session will be conducted by Professor Erastus Amayo, a neurologist with the University of Nairobi and an expert on *T. solium* cysticercosis. A draft schedule is as follows:

Day 1

- Health Education
- Discussion on Epilepsy
- Discussion on Cysticercosis in general
  - Transmission cycle
  - Porcine cysticercosis
  - Neurocysticercosis
- Risk factors for acquiring cysticercosis – hygiene and sanitation, pig management practices etc

Day 2

- Clinical presentation
- Diagnosis and Treatment
- How to manage complications during treatment

At the end of Day 2, the groups may be split up and a separate session will be held for Lab technicians in order to ensure that the sera collected is collected properly and stored and labelled in the correct fashion.

The epileptic patients who have a confirmed diagnosis either by CT scan or serology will then be referred to the trained clinicians for management of their condition and the drugs necessary for treatment will be funded by the University of Edinburgh. It is important to note that the sera collected from the patients will not be tested for HIV.

15. Expected Benefits

The benefit of the study to the respondents would be an affirmative diagnosis for neurocysticercosis, if present by either serology or CT scan, thus enabling those afflicted to receive treatment. According to the diagnostic criterion set out in "The Current Consensus Guidelines for the Treatment of Neurocysticercosis" (Garcia 2002), patients who exhibit cystic lesions showing the scolex of the parasite on a CT scan or an MRI fit the "absolute" criterion. The presence of cysts in the brain indicates a definitive diagnosis.

Definitive diagnosis – these are patients who have one absolute criterion or those who have two major plus one minor and one epidemiologic criterion, or
Probable diagnosis – those patients who have one major plus two minor, those who have one major plus one minor and one epidemiologic condition, and those patients who have three minor plus one epidemiologic condition (Del Brutto 2001).

There is no standard regimen for the treatment of neurocysticercosis but the treatment varies with the type of involvement and the presence of other factors. Management of neurocysticercosis includes the use of symptomatic medication (including anticonvulsants), anti-inflammatory drugs, anti-parasitic drugs, or surgery (Murrell 2005). The table below illustrates the criteria guiding clinical management of the disease.

Figure 5: Treatment Management Guidelines for Cysticercosis

	Parenchymal neurocysticercosis
Viable (live cysts)	
One to five cysts	Anti-parasitic treatment, with corticosteroids
More than five cysts	Anti-parasitic treatment, with corticosteroids
	Anti-parasitic treatment, with high-dose corticosteroids. Alternatively, chronic steroid management. No anti-parasitic treatment, neuro-imaging follow-up
Enhancing lesions (degenerating cysts)	
Mild or moderate	No anti-parasitic treatment. Neuroimaging follow-up. Alternatively anti-parasitic treatment with corticosteroids and as a second alternative anti-parasitic treatment. Corticosteroids only if side effects develop
Heavy (cysticercotic encephalitis)	No anti-parasitic treatment. High dose corticosteroids and osmotic diuretics
Calcified cysticerci	
Any number	No anti-parasitic treatment
	Extraparenchymal neurocysticercosis
Ventricular cysticercosis	Neuroendoscopical removal, when available. If not available then CSF diversion followed by an anti-parasitic treatment with corticosteroids, and as a second alternative, open surgery (mainly for cysts in the fourth ventricle)
Subarachnoid cysts including giant cysts or "racemose" cysts on neuroimaging	Anti-parasitic treatment, with corticosteroids. Ventricular shunt if there is hydrocephalus
Hydrocephalus with no visible cysts on neuroimaging	Ventricular shunt. No anti-parasitic treatment
Other locations	
Spinal cysticercosis, intra or extramedullary	Primarily surgical. Anecdotal reports of successful use of albendazole with corticosteroids
Ophthalmic cysticercosis	Surgical resection of cysts

Source: (Murrell 2005)

16. Expected Benefits to General Population

The expected benefits to the general population will be much more knowledge gained on porcine cysticercosis, epilepsy and epilepsy acquired from *Taenia solium* (NCC). In addition, pig husbandry practices designed to limit the risk of continuing the cycle of cysticercosis will have been explained to the farmers in the context of the two-day workshop to be held for District Health personnel and others.

17. Involvement of Research Subjects with Limited Capacity

In the event that a research subject is deemed to have limited capacity, a suitable guardian will assist in the obtaining of informed consent and all care will be taken to minimize risks and discomfort to such subjects. The justification for involving a vulnerable subject would be that they have been shown to have a diagnosis of epilepsy and that the knowledge that their epilepsy is acquired from exposure to *T. solium* and therefore subsequent potential for treatment would outweigh the discomforts in obtaining the diagnosis.

18. Individual Informed Consent

The informed consent for serological sampling for *Taenia solium* will be requested in English, Kiswahili or the local language. It will be translated by the enumerators and the respondent will reiterate the consent in the presence of a witness to confirm comprehension. The witness, enumerator and respondent patient will sign the form to confirm the context of the respondent's participation in this study has been understood by all parties. In the case of a subject to be sampled who is considered a minor (under 16), a parent or legal guardian will be required to give consent. Additionally, the minor will be asked for oral consent. In cases in which participants are illiterate, the form will be read to them and the understanding of its content will be ensured. The witness will be selected by the patient and will only sign the form when s/he is convinced that the patient has understood the objectives of the study, the sampling procedure as well as the risks, rights and benefits involved in the study.

All participants involved in the collection of serology and accompanying questions will be required to sign an Informed Consent Form (Appendix V). There will be a

subsequent Informed Consent Form (CT scan) for those participants who are to undergo a CT scan (Appendix VI).

19. Information Arising From the Study on Harm, Benefit and Other Research That Could Affect Participation

All information concerning benefits and risks to participants including that from other studies will be communicated to the subjects at the time of obtaining informed consent. It is not anticipated, however, that this issue is pertinent.

20. Plans to Inform Subjects Ultimately About the Results of the Study

As medical personnel in Busia District are already participating in the serology collection, CT scans and subsequent follow-up treatment, they will assist in informing participants of the results of the study.

21. Confidentiality of Personal Data Including Subjects' Privacy

The patients' information is considered confidential and to be used only for purposes of this study. Patients will be assigned a unique identifying number and information concerning their status with respect to neurocysticercosis, taeniosis and exposure or lack of exposure to *T. solium* will be reported using only this identifier. All enumerators and technicians are required to sign a confidentiality agreement (Appendix VII) acknowledging the patients' rights to protect their privacy. There are no genetic results being sought in this study.

22. Further Uses for Research Materials

While it is not immediately foreseen that the biological samples will be used for further research, should a possibility arise for using these biological samples to enhance the control of cysticercosis, further the objectives of the study, or improve diagnostics, permission has been sought in the Informed Consent for Serology (Appendix V) The research results pertaining to information sought about risk factors for exposure to *T. solium* (Appendix II) may be used in comparison with similar studies, for the purpose of enhancing general knowledge regarding the control of the disease.

23. Plans for Statistical Analysis

The study will be analysed using standardized logistical regression techniques (Woodward 1999), (Dohoo 2003), (Thrusfield 2005) and will suppose that all assumptions in these techniques remain constant. The statistical power will be calculated at 80% as the sample size is statistically significant and the data that previously exists concerning prevalence rates for neurocysticercosis supports the hypothesis that similar rates will be found in this study. The study will be terminated if the sample size reaches below 350 tested based on an exposure rate of 3% for controls, 10% for cases (95% CI and power of 80%).

24. Sponsor Organization

The University of Edinburgh
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&

The International Livestock Research Institute
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Nairobi, Kenya

Both of these institutions have a long working history together. The investigators have been previously cited and their curriculum vitaes included as Appendix I.

25. Source and Funding

The source of the funding for the research:

UK Department for International Development

Animal Health Program

AHP No: AHP-BO-05/05

Project Title:

Assessing the burden of cysticercosis, with relevance to both human health and livestock production economics, on smallholder communities in western Kenya.

The amount of the award is £20,000. (See Appendix VIII for grant information).

26. Conflict of Interest

There are no conflicts of interest.

27. Adequate Facilities

Biological materials will be stored at the International Livestock Research Institute in Nairobi, prior to being sent to a laboratory in Belgium for analysis. This laboratory is housed within the Prince Leopold Institute for Tropical Medicine in Antwerp. The sera will be analysed using an antigen detecting ELISA which has reported a sensitivity rate of 85% and a specificity of 92% (Garcia, Parkhouse *et al.* 2000). A study done in Vietnam evaluating the Ag ELISA found a very high agreement between an ELISA for detecting circulating antigen, computerised tomography scanning and biopsy examination of subcutaneous cysticerci (Erhart 2002). The analysis of the samples will be performed under the supervision of Professor Pierre Dorny, Department of Animal Health, Institute of Tropical Medicine in Antwerp, Belgium. Depending on the results of this diagnostic test, further diagnostic tests may need to be carried out to better diagnose cysticercosis or taeniasis.

28. Declaration of Helsinki and CIOMS Guidelines

All principles set out in the Declaration of Helsinki and the Council for International Organisations of Medical Sciences (CIOMS) International Ethical Guidelines for Biomedical Research Involving Human Subjects will be strictly adhered to and implemented.

29. Code for Subjects' Identities

The code for the subjects' identities will be assigned by the principal investigator and stored separately from the responses to the survey. Individual responses will

thereafter be referred to by codes alone. The name of the subject and the names of household members will not be shared with individuals outside the survey organization and the identifying information linking names to codes will be destroyed once the information is compiled into a computer. The code can be broken either by the principal investigator or by Professor Sue Welburn, whose contact details appear in Appendix I in the event of an emergency.

30. Time schedule

Serology data collection will take place in the month of December 2006, the samples then sent for analysis during January and February of 2007 and the CT scans will take place at the end of February or the beginning of March 2007. The final write-up and results of the study will be published by June 2007.

31. Instructions for staff involved in the trial

All enumerators, clinical officers, drivers and others involved in the study will be asked to sign a confidentiality agreement guaranteeing anonymity for the study participants (Appendix VII) and will attend a training course prior to serum collection and analysis.

32. Ethical Issues

The investigators are fully aware of all ethical issues pertaining to this study and believe that the benefits to the patients obtaining a diagnosis with respect to their epilepsy and therefore the occasion to treat it, clearly outweighs any of the anticipated risks. The participants in the study are volunteers who are fully informed and will derive no benefit other than an opportunity to improve their health status. The participants' integrity and rights to privacy have been taken into consideration and all reasonable care is being taken to ensure confidentiality of data. The participants have all been informed of their right to withdraw from the study at any time with no punitive action being taken against them or their families. All care will be taken to preserve the accuracy of the results of this study and negative as well as positive results will be published and publicly available.

33. Rights to Publish

As the study is being funded by the DFID through University of Edinburgh, this institute holds the data. The principal investigator owns the results of the study, the raw data and any analysis of that data can only be done with the express permission of the principal investigator. This study is a part of the principal investigator's PhD research and will be published by that individual, although all collaborators who make a significant contribution to the scientific development of the project will be given the opportunity to participate in the publication process.

34. Proposed Research Summary

The aim of this proposed research is to determine how many people out of a population of epileptics in Busia District have epilepsy as a result of exposure to pork tapeworm (*Taenia solium*). This will be determined using patients' blood and testing it to see if there are any parasite antigens present and also by a CT scan which will scan the brain to see if there are any cysts present. In this way, we will be able to have an idea of the prevalence of epilepsy caused by the pork tapeworm in Busia District. In addition, we would like to see if there are any common practices, conditions or habits in the way that they live and in the way in which they keep pigs amongst the people who have been exposed to the tapeworm which may be contributing factors to their exposure. These common practices, conditions or habits are called risk factors. We will ask the participants in the study a series of questions about how they manage their pigs, their hygiene practices and other factors that are of interest.

Once the data about the risk factors has been collected and their status with regard to exposure to the tapeworm has been established, we will be able to draw conclusions about contributing risk factors.

35. References

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Appendix I: Curriculum Vitaes of Investigators

Katharine Downie

Date of Birth	15th August 1967
Nationality	Canadian/British
Address	P.O. Box 30709 00100, Nairobi, Kenya
	Tel: +254 (0) 733-512-562(mobile)
	Email: k.downie-ngini@cgiar.org

Programme Management

My programme management experience is in public health and water and sanitation. At the African Medical and Research Foundation (AMREF), I was in charge of the administrative and financial aspects of grant management for 2 projects in different areas of Somalia a project in Luuq, Somalia and one in North West Galgaduud, Somalia, both funded by the European Community Humanitarian Office (ECHO). The projects involved management of a team of health care workers - physicians, nurses and auxiliary health personnel, running Primary Health Care (PHC) Centres in these districts. My responsibilities included developing job descriptions; conducting training; financial management; report writing, and proposal writing.

Financial Management

My consultancy company (K.D Consulting Limited) was contracted to provide financial management to Maji na Ufanisi (formerly WaterAid UK). My role included financial management of the institution and its projects. I was responsible for all financial reporting to the donors, development of budgets and development of financial reporting and budgeting systems. My tasks also included all proposal writing and supervision of administrative and technical staff.

Research Experience

My primary area of research has been zoonotic disease, in particular, the human health impact of *Taenia solium* neurocysticercosis. The study has involved determining the burden of this condition using DALYs as well as the livestock economic losses associated with affected swine. Risk factors such as pig husbandry practices, hygiene and sanitation practices and poverty have been taken into consideration as contributors to neurocysticercosis-acquired epilepsy. Intervention strategies for prevention of the condition have also been introduced.

EDUCATION

2003 - present	PhD - Tropical Animal Health Centre for Tropical Veterinary Medicine Royal (Dick) School of Veterinary Studies The University of Edinburgh Easter Bush Roslin, Midlothian Scotland *(Ongoing PhD studies in Kenya (ILRI Graduate Fellow) - expect completion by December 2006)*
1987 - 1991	Bachelor of Arts English Literature *Minor - Gender Studies* McGill University, Montreal, Canada
1985 - 1987	Diplôme de Niveau 1 L'Institut Catholique de Paris Paris, France *Obtained highest level diploma in French language fluency*
1980 - 1985	Ontario Secondary School Honours Graduation Diploma Havergal College, Toronto, Canada *Received Ontario Scholarship, Politics and Literature Prizes*

WORK EXPERIENCE

June 2002 - October 2003

International Livestock Research Institute (ILRI)
Naivasha Road, PO Box 30709 00100, Nairobi, Kenya

Special Assistant to the Director General

For this period I provided consultancy services to ILRI in the capacity of the Special Assistant to the Director General. The position demands managing the Institute Management Committee, writing research papers, speeches, concept notes, PowerPoint presentations and other documents as the Director General requires. It necessitates an in depth knowledge of development issues and the instruments by which poverty alleviation can be achieved. It also entails managing the budgets for the office, supervising staff and travelling with the Director General as necessary. In 2002-2003, ILRI underwent an organisational change and restructuring. During this period I was involved in the development of various documents, including **"Livestock, a pathway out of poverty - ILRI's strategy to 2010".**

August 2001 - present

KJD Consulting Limited
PJ Place, Enterprise Road, Industrial Area, Nairobi, Kenya

Managing Director

KJD Consulting Limited expertise is based largely on experience gained by working in the Livestock, Public Health and Development arenas over the past 15 years. Consultancy jobs range from proposal writing, monitoring and evaluation of projects to financial reporting to donors.

Have started own consultancy business focusing on financial management, business plan writing for private individuals and businesses for venture capitalists or other interested investors, proposal writing for NGOs and Community based organisations, strategic plan development and use of other business planning tools for both public and private sector. In addition, consultancies have been undertaken performing analysis on various development tools such as Participatory Urban Analysis, Baseline Surveys and other Monitoring and Evaluation Impact Assessment strategies.

July 1999 - July 2001

Water and Development/Maji na Ufanisi
Chiromo Access Road, Nairobi, Kenya

Finance Administrator

Responsibilities
- financial management of institution and its projects (donors include DFID, SIDA, Oxfam, WaterAid and other smaller ones)
- development of sound financial systems
- supervision and development of budgets
- preparation of financial reports including
  - monthly reports
  - quarterly reports
  - final annual reports
  - donor reports as required
- supervision of administrative staff
- administration of the Nairobi office
- all proposal writing within the organisation
- editing and writing of all narrative donor reports

March 1994 - June 1999

African Medical and Research Foundation (AMREF)
Wilson Airport, Nairobi, Kenya

Programme Officer/Project Manager - Somalia Projects
Nomadic Health Unit
Clinical Department

This job required an in-depth knowledge of Public Health and Development issues to be capable of writing comprehensive proposals for various donors.

Responsibilities
- Co-ordination and financial management of two projects dealing with Primary Health Care funded by the European Union in Luuq and NW Galgaduud Districts, Somalia;
- Developing financial systems to ensure accurate knowledge of project expenditure accruals, projected expenditure and final expenditure meeting specific conditions from donors.
- Writing project proposals to secure funding including drawing up

- Preparing financial and narrative reports in compliance with donor requirements (this involved developing new accounting procedures as AMREF was not familiar with EU reporting);
- Writing workshop and seminar proceedings, annual reports, workplans;
- Corresponding with national offices and donors;
- Assuming financial responsibility for unit funds and operation of two project bank accounts;
- Conducting training sessions on Primary Health Care (PHC) and the development of job descriptions for use in Somalia;
- Acting Programme Co-ordinator when necessary
- Member of the *Somalia NGO Consortium* and *Somalia Health Co-ordination Group*

August 1993 - March 1994

Senior Logistics Officer - Somalia Project
Nomadic Health Unit
Clinical Department

Responsibilities
- Co-ordinating supplies between Nairobi and project site in Somalia;
- Implementing inventory and stock systems;
- Compiling quarterly expenditure reports for donors based on supply acquisition.

January 1993 - August 1993

Research Officer
Women and Development Unit
Community Health Department

Responsibilities
- Assisted in the development of AMREF's gender policy;
- Conducted a survey of health related NGOs to establish an inventory of NGOs, government departments and donor agencies dealing with women's health and development.

1991 - 1992

Canadian Executive Service Organisation (CESO)
Toronto, Canada

Project Administrator (Africa, Asia, Caribbean)

Responsibilities
- Co-ordinating the details of project volunteers' overseas assignments;
- Liaising with the client in the project country and the volunteer;
- Contributing editor to the monthly *CESO News Bulletin.*

Liaison - Women and Development Programme

Responsibilities
- Assisted in the development of the mission statement and policy objectives of CESO's Women and

- Development (WID) programme;
- Member of the Women and Development Steering Committee;

CONFERENCES AND WORKSHOPS ATTENDED/ORGANIZED

15th - 18th June 2005

Zoonoses: From Science to Policy
Organised by the Health Protection Authority, Liverpool, UK
- Participated under the auspices of the University of Edinburgh in a conference which examined translating policy frameworks and modelling concerning zoonoses control in a developed country setting

21st - 25th April 1997

Training/Continuing Education Working Group Workshop: *Towards a National Training Programme for Somalia*
Nairobi
- Co-organised and co-facilitated this workshop which aimed to develop a training plan for Somali health workers and to ratify job descriptions for different cadres of Primary Health Care workers
- Developed job descriptions for different cadres of Primary Health Care service providers

March 1996

Project Cycle Management: Developing a logical framework (European Union)
- Development of projects using tools such as *stakeholders' analysis, logical framework, baseline survey and participatory rural appraisal*

16th - 17th May 1995

UNICEF Somalia Health Strategy Workshop
Nairobi
- Was a member of both Essential Drugs and Training Curricula development working groups;
- Developed Plan of Action for Training Curricula development in Somalia

27th June - 2nd July 1994

Workshop to Develop the 1995-1997 Luuq District Hospital Strategic Plan and Operational Plan
Luuq, Gedo Region, Somalia
- Responsible for organisation of workshop and production of final workshop document;

DOCUMENTS PRODUCED

Training/Continuing Education Workshop Report: Towards a National Training Programme for Somalia. Downie, Katie and Lucy Wood, April 1997.

Gedo Region Workshop Report. Nomadic Health Unit, April 1994.

Rational Use of Drugs, MCH and OPD Drug Lists and revised reporting formats. UNICEF Somalia, January 1995.

Luuq District Hospital Strategic and Operational Plans 1995-1997, July 1994

CONSULTANCIES

Japan International Cooperation Agency ***(JICA)***
Nairobi, Kenya
Rapporteur

Asia-Africa Knowledge Co-Creation Program (AAKCP) Rural Development Sub-Program (RCDS)
Final Seminar
Responsible for producing the Proceedings for the Asia-Africa Final Seminar
Held at AICAD, Jomo Kenyatta University of Agriculture and Technology, Juja, Kenya
27th - 29th June 2006

Rural Focus Limited
Nanyuki, Kenya

Editing:
Report on the Operationalisation of the Water Act 2002 in Water Resources Management, *Republic of Kenya, Ministry of Water and Irrigation* in collaboration with the ***Water Resources Management Authority***, October 2005. Supported by the Swedish International Development Cooperation Agency (Sida)

Maji na Ufanisi/Water and Development

Analysis of Nairobi Slum Programme Participatory Urban Analyses -August 2001

Maji na Ufanisi/Water and Development

Staff Rules and Regulations: An Operational Manual

ISP Kenya Ltd **ISP Kenya Ltd - A Business Proposal - January 2001**
ISP Kenya Ltd MailAfrica International - A Business Proposal - January 2001
ISP Kenya Ltd ISP Kenya Ltd and MailAfrica International - Five Year Business Plan for Strategic Investment - January 2001

Maji na Ufanisi/Water and Development

Proposal to Ford Foundation - Nairobi Slum Programme: *Building Democratic Institutions and Community Assets within the Slums around Nairobi* - September 2000

Maji na Ufanisi/Water and Development — Proposal to CORDAID/SIDA - Nairobi Slum Programme: Developing Water and Environmental Sanitation Through Community- Based Organisations - A three-year proposal - April 2000-March 2003 - March 2000

Maji na Ufanisi/Water and Development — **Proposal to DFID (Department for International Development (UK) Developing ASAL Water Through Local Organisations - A three year proposal April 2000-March 2003** - April 2000

SIDA Quarterly Report - *Capacity building of community-based organisations to improve the management of access to safe water and sanitation in Nairobi slums.* **Maji na Ufanisi (Water and Development Kenya)**.March 1999.

Developing Arid and Semi-Arid Lands' Access to Water Through Local Organizations. Proposal for funding for **Maji na Ufanisi (WaterAid Kenya)** to the European Union. January 1999.

Rapporteur Health and Nutrition Workshop - Rational Use of Drugs, MCH and OPD Drug Lists and Revised Reporting Formats. **UNICEF Somalia**, 18 - 22 November 1994.
- Responsible for production of minutes and draft document.

ASSISTED IN THE PREPARATION OF

Building Technical Capacity and Gender Advocacy in Young Professional African Women. Conference Proceedings, ICIPE Science Press, Nairobi, July 1993.

Luuq District Hospital Master Plan 1995-1997 and Operational Plan 1995. Nomadic Health Unit, July 1994.

Tangulbei Division Health and Health Care Inventory and Feasibility Study, Oyaya, Charles, Heering, N. J., Begley, Fr. Michael G., King, Basil, February 1994.

OTHER SKILLS

Languages English - fluent spoken and written; French - fluent spoken; German - medium proficiency spoken; Swahili - medium proficiency spoken

Computer Proficiency: Microsoft Word; Microsoft Excel; Microsoft PowerPoint; Microsoft Access; DBASE III, IV; Corel 9

Curriculum Vitae

Susan Christina Welburn

Professor of Medical and Veterinary Molecular Epidemiology
Centre for Infectious Diseases
College of Medicine and Veterinary Medicine
University of Edinburgh
EH25 9RH
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University education

1988–1991	PhD, Faculty of Medicine, University of Bristol
1980–1984	BSc, University of the West of England, Bristol

Degrees awarded

1991	PhD, Faculty of Medicine, University of Bristol '*The Rickettsia-like organisms of Glossina ssp.*'
1984	BSc Applied Biological Science, Class 2.1 Hons.

Career since graduation

2006 -	Professor of Medical and Veterinary Molecular Epidemiology, Centre of Infectious Diseases, Royal (Dick) School of Veterinary Studies, College of Medicine and Veterinary Medicine,
2002 – 2006	Reader, Centre of Infectious Diseases, Royal (Dick) School of Veterinary Studies, College of Medicine and Veterinary Medicine, The University of Edinburgh.
1999 - 2002	Senior Research Fellow (Research Grade AR3), Centre for Tropical Veterinary Medicine, Division of Tropical Animal Health, Royal (Dick) School of Veterinary Studies, The University of Edinburgh.
1998 - 1999	Visiting Scientist, International Livestock Research Institute, Nairobi, Kenya.
1996 – 2000	Wellcome Trust Career Development Fellow, Centre for Tropical Veterinary Medicine, Royal (Dick) School of Veterinary Studies, The University of Edinburgh, Easter Bush, Roslin, UK & previously Division of Molecular Genetics, University of Glasgow, Anderson College, 56 Dumbarton Road, Glasgow G11 6NU.
1991 - 1996	Post-Doctoral Research Assistant, Tsetse Research Group, School of Veterinary Science, University of Bristol, Langford, Bristol.
1986 – 1991	Research Assistant, Tsetse Research Group, School of Veterinary Science, University of Bristol, Langford, Bristol.
1984 – 1986	Research Associate, Tsetse Research Group, School of Veterinary Science, University of Bristol, Langford, Bristol.

Major research interests

My research interests have focused on understanding the interactions between parasites and their vectors and hosts that lead to transmission of human disease. Studies follow four lines of research, epidemiology of human sleeping sickness, zoonotic infectious disease (sleeping sickness, rabies, cysticercosis, brucellosis and bovine TB) and transgenic insect control. This involves a dissection of the mechanisms of innate resistance of vectors to parasite infections and the complex interactions between host, vector and parasite that result in parasite differentiation and disease transmission. Research has resulted in over 100 peer reviewed

publications to date including 7 book chapters, 18 peer refereed reviews and original peer reviewed papers.

Principal research grants awarded

Title of project	Funding body	Duration	Award £
Using e-learning to build capacity for in-country delivery of medical and healthcare professional education in Malawi (Co-PI)	Scottish Executive	2006-2008	**176,000**
E-learning networks for Asia and Africa (With Prof Pfeiffer, Dr Eisler)	DFID	2006-2009	**250,000**
Roll back sleeping sickness campaign phase I (PI with Dr Eisler).	CEVA/IK	2006-2007	**350,000**
Assessment of overlap between *T.b.rhodesiene* and *T.b.gambiense* sleeping sickness in Uganda (PI)	World Health Organization.	2006-2007	**12,000**
Assessment of optimal channels of communication for effective message delivery on nagana and human sleeping sickness in Uganda (PI).	Wellcome Trust	2006-2009	**120,928**
Enabling e-distance learning in International Animal Heath (PI with Dr Eisler)	Commonwealth Scholarship commission	2006-2009	**130,000**
University Shared scholarship scheme (MSc) – Miss Pratij Raj	DFID Shared scholarship scheme	2006-2007	15,000
Development of Masters programme in International Animal Health (Co-PI)	EU (Principals e-learning fund)	2005-2007	**40,000**
Methods for control of zoonotic Sleeping sickness in SE Uganda. (PI)	DFID	2005-2006	**263,366**
Domestic animals as reservoirs of *T. b. gambiense* in S. Sudan (Co-PI)	USAid	2005-2006	13,000
Cistercercosis and human health in W. Kenya. (Co-PI)	DFID/ AHP	2005-2006	30,000
Pigs as a reservoir for human sleeping sickness (Co-PI)	DFID/ AHP	2005-2006	20,000
MRC studentship – Harriet Auty	MRC	2004-2007	**50,000**
Refining and extending DALY calculations for sleeping sickness. (PI)	World Health Organization.	2003-2005	25,000
Impact assessment of FITCA intervention trials in SE Uganda (PI)	EU/DFID	2004-2005	**45,000**
University Shared scholarship scheme (MSc) – Miss Gudeep Lall	DFID Shared scholarship	2004-2005	15,000
Decision support system for the control of Sleeping sickness in SE Uganda. (PI)	DFID	2002-2004	**150,000**
Symbiosis – Infection without disease.	Wellcome Trust	2002-2005	**175,454**
Sleeping sickness surveillance W. Nile (PI)	World Health Organization	2002-2004	15,000
Centre for infectious Diseases Studentship - Jenna Fyfe	CID/UOE	2002-2005	**50,000**

Development of a novel technique for differentiating *Trypanosoma brucei* parasites (PI)	Cunningham Trust	2002-2005	**66,434**
The Impact of Control Products on Insect Populations: A Mathematical Approach ('In-Model') (Co-PI)	Edinburgh Technology Fund.	2002-2003	14,700
Development and incorporation of Bacteriophages as pest control agents (Co-PI)	SMART Scotland	2002-2003	**78,272**
Variant surface glycoproteins expressed by *Trypansoma brucei* in endemic areas (Co-PI)	Leverhulme Trust	2001-2004	**97,459**
Bacteriophage - next generation insecticides (PI)	Edinburgh Technology Fund	2001-2002	**104,914**
Decision support system for the control of Sleeping sickness in SE Uganda (PI)	DFID	2000-2003	**349,827**
Isolation and characterization of genes encoding secretory proteins of the ixodid tick (Co-PI)	Leverhulme Trust	2000-2003	**97,165**
Molecular analysis of programmed cell death and differentiation in *T. b. rhodesiense* (Fellowship – 4 year Career Development Award)	Wellcome Trust	1996-2000	**255,000**
DNA sequencer University of Glasgow - Research Equipment Initiative	Wellcome Trust/ MRC	1997	**97,010**
The role of lectins in the transmission of trypanosomes in tsetse flies.	Wellcome Trust	1991-1995	**140,271**
TOTAL			**3,246,800**

Research status:

My research was included as International. I have published over 90 primary research papers, the most significant for RAE being **three publications in the Lancet and one in The British Medical Journal on the molecular epidemiology of sleeping sickness and role of domestic livestock in sleeping sickness epidemiology and one in PNAS on molecular evolution of pathogenesis in symbionts**. I have also had eleven peer reviewed review articles published in the past 5 years including **6 in Trends Journals and 1 in Lancet Infectious Diseases**. I have been successful in the acquisition of over **2,578,519** million pounds of research funding since 2001.

Research supervision experience:

Postdoctoral training - I have mentored seven post doctoral staff members: Dr Colin Dale (now working US); Dr Kevin Arnold (now working US); Dr Alistair Darby (now working in Sweden); Dr Aimee Tilley; Dr Eric Fevre; Dr Simon Young; Dr Euan Macloed, Dr Kim Picozzi.

Postgraduate Research training - 16 completed PhD and 2 completed MSc students.

Colin Dale (1997). *The secondary (S) Symbionts of Glossina.* PhD University of Liverpool - 2nd supervisor.

Simon Lillico (1999). *Characterisation of genes identified during a RADES-PCR screen of concanavalin A – treated procyclic Trypanosoma brucei rhodesiense.* PhD University of Glasgow - Principal supervisor.

Simon Young (2002). *Genetic features of Sodalis glossinidus - a symbiont bacterium of tsetse flies'.* PhD University of Glasgow - Principal supervisor.

Eric Fevre (2002). *The epidemiology of trypanosomiasis, a re-emerging zoonosis in Uganda'.* PhD University of Edinburgh - 2nd supervisor.

Martin Odiit (2003). *Epidemiology of Trypanosoma brucei rhodesiense sleeping sickness in Eastern Uganda.* PhD University of Edinburgh - 2nd supervisor

Magai Kaare (2004). *Sleeping sickness in the Serengeti ecosystem'* MSc Sokoine University, Tanzania - 2nd supervisor

Karine Delroux (2004). *Cloning of genes expressed in the salivary glands during feeding of the tick Amblyoma variegatum.* - PhD University of Edinburgh - 2nd supervisor

Noreen Machila (2005). *Improved targeting and appropriate use of trypanocidal drugs for the control of African bovine trypanosomiasis in tsetse endemic areas of western and coastal Kenya within the context of primary veterinary care.* PhD University of Edinburgh - Principal supervisor.

Christine Thuranira (2005). *Socio economic factors influencing livestock keeping dynamics on a smallholder crop-livestock system in Western Kenya.* PhD University of Edinburgh - Principal supervisor.

Gurdeep Lall (2005). *Development of amplified fragment length polymorphism (AFLP) marker analysis for the study of Glossina pallidipes population genetics'.* MSc University of Edinburgh - Principal supervisor.

Ewan Macloed (2005*). Factors affecting transmission of trypanosomes through tsetse flies* PhD University of Edinburgh - Principal supervisor.

Hachemi Zerria (2006) *Molecular analysis of insect stage Trypanosoma brucei.* . PhD University of Edinburgh - Principal Supervisor.

Francis McOdimba (2006) *Competition between T. brucei in domestic livestock in S.E Uganda'.* - PhD University of Edinburgh - 2nd supervisor.

Olga Tosas (2002–2005). *Tick borne disease interactions in cattle in East Africa.* University of Edinburgh - 2nd supervisor

Andrew Cox (2003– 2006) *Development of molecular tools for the diagnosis of trypanosomiasis'* University of Edinburgh - Principal supervisor.

Caroline Mathews (2003–2006). *The mechanisms of Sodalis glossinidius symbiosis in tsetse flies'* University of Edinburgh - Principal supervisor.

Magai Kaare (2003–2006). *Assessment of wild animals in cycle of rabies transmission in the Serengeti*. University of Edinburgh - 2nd supervisor

Tziana Lembo (2003-2006) *Genetic diversity of circulating Rabies strains in Tanzania.* University of Edinburgh – 2nd supervisor.

Ongoing postgraduate supervision -*Involvement in postgraduate training of sixteen registered postgraduate students at the University of Edinburgh (including nine veterinarians and one medic)*

Beatrix Wisseman (2003- 2006). Zoonotic *disease transmission in sleeping sickness foci in East Africa.* University of Edinburgh - Principal supervisor

Jenna Fyfe (2003– 2006). *Interventions for control of human sleeping sickness.* University of Edinburgh - Principal supervisor.

Andrew Brownlow (2003–2006). *Restricted application of insecticides on cattle and impact on animal health.* University of Edinburgh - 2nd supervisor

Neil Anderson (2004-2007) *Wildlife as reservoir of human and animal trypanosomiasis Luangwa Valley, Zambia.* University of Edinburgh - Principal supervisor.

Joseph Mubanga (2004 - 2007). *Trypanosomiasis and animal health in Zambia.* University of Edinburgh - 2nd supervisor.

Kohei Makita (2004 - 2007). *Peri-urban livestock, human and animal disease transmission.* University of Edinburgh - Prinicipal supervisor.

Harriey Auty (2004–2007). *Reservoirs of human sleeping sickness in Serengeti wild life.* University of Edinburgh - Principal supervisor

Lucas Matamba (2004–2007). *Human Health and sleeping sickness in Tanzania.* University of Edinburgh - Principal supervisor

Katie Downie (2004–2007). *Cysticercosis pig keeping and impact on human health.* University of Edinburgh - Principal supervisor

Heba Ahmed (2005 - 2008). *Competition between T. brucei subspecies and impact on epidemiology of trypanosomiasis in East Africa.* University of Edinburgh - Principal supervisor

Raj Prashanti (2006-2007). MSc. *Insects as environmental sensing tools.* Principal supervisor

Ceasar Lubaba (2006-2009). Mixed infection disease modelling in East African cattle – 2nd supervisor

Wandee Kongkaew (2006-2009). Risk analysis for Avian Influenza in Thailand – 2nd supervisor.

Ayo O Majekodunmi (2006- 2009). Sleeping sickness and animal trypanosomiaisis in Nigeria Principal supervisor

Nichola Batchelor (2006- 2009). GIS informatics and human sleeping sickness. Principal supervisor.

Teaching

As a Professor in the Centre for Infectious Diseases I teach on our Tropical Animal Health Elective to final year students. This involves lectures on aspects of Tropical Zoonotic Medicine, principally zoonotic and infectious diseases. I supervise 2 honours students per year for the Medical Microbiology Honours course. I also offer a short course option on trypanosomatid diseases of animals and humans for the Medical Microbiology course. I also participate in the Medical Microbiology seminar series for Final Year students. Chair examining boards for distance learning Masters Programme in International Animal Health and Infectious Diseases (3 year e-distance learning master's course). Awarded GBP 130,000 in scholarship and training e-tutors in Makerere Vet School. Learning materials developed for this course will feed into our Tropical Animal Health and other electives for the Vet Students and add to the Medical Microbiology programme. I run various Transferable (Transkills) option courses for postgraduate students on report writing and thesis preparation and participate in Wellcome sessions for new postgraduate students.

Administration

2000 -	GMSO representative Easter Bush Vet Site
2004 -	Biological safety officer Easter Bush Site.
2002 -	Company Director In-Phage - University of Edinburgh spin-out
2000 - 2004	Treasurer British Society for Parasitology
2001-	Postgraduate representative for Animal Health and Welfare - divisional representative for all student progress assessments.

Membership of societies where academic distinction is the criterion of membership

2006 -	Council, British Society of Parasitology
2005 -	Member steering committee of AUVEC
2000-2004	Treasurer British Society for Parasitology
1991 -	Member British Society of Parasitology

Membership of committees relevant to research

2005 -	Management committee Royal Zoological Society Edinburgh for Research and Conservation activities of Budongo Forest, Uganda
2005 -	Organizer British Society Parasitology Autumn Symposium, Linnean Society, London.
2005 -	Member, Steering Committee Distance Learning Initiative, University of Edinburgh
2004 -	Member, Organizing committee 31st Trypanosomiasis and Leishmaniaisis Seminar
2002 -	Meeting organizer 30th Trypanosomiasis and Leishmaniasis Seminar

2002 - Member, Research and Postgraduate Committee.

2002 - Divisional Postgraduate representative, AHW.

2001 - 2004 Treasurer British Society for Parasitology

1999 - Member, Veterinary Faculty Post Graduate Committee

1999 - Member, Research Committee, Equipment Committee

International Scientific Meetings organized:

2005 - Programmed Cell Death and the Protozoan Parasite, Autumn Symposium, British Society of Parasitology and the Linean Society London (Organizer)

2005 - Capacity building for the African animal health sector: addressing the need for new learning opportunities. ICPTV/DFID, Naivasha, Kenya (Organizing Committee)

2004 - 31st Trypanosomiasis and Leishmaniasis Symposium, British Society of Parasitology,
Ceske Budovice. Czech Republic (Organizing committee)

2002 - 30th Trypanosomiasis and Leishmaniasis Symposium, British Society of Parasitology, Edinburgh (Meeting organizer)

Reviewer for the following journals: Nature Genetics, British Medical Journal, FEMS Microbiology Letters; Microbiology, Trends in Microbiology, Trends in Parasitology, Trends in Microbiology, Acta Tropica, Parasitology, Experimental Parasitology. PLOS Medicine

Reviewer for the following grant awarding bodies: Wellcome Trust, BBSRC, MRC, and National Institutes of Health, EU.

Items of esteem at International symposia and congresses.

2005 Invited Speaker. Cell death and differentiation – Death and Differentiation of trypanosome infections in tsetse – Cell Death and the Protozoan Parasite. November 2005. The Linnean Society, London.

2005 Invited Speaker. Control of Zoonotic Disease – a route to poverty alleviation .World Health Organization, 20 -21 September, 2005, Geneva, Switzerland.

2004 Invited speaker. Fitness costs associated with resistance to human serum in *Trypanosoma brucei rhodesiense*: theoretical predictions and field data. 31st Trypanosomiasis and Leishmaniasis Symposium, Cezske Budowize, Czech Republic.

2003 Invited Speaker. Control of sleeping sickness in domestic and world animal reservoirs. World Parks Congress, ICUN, September 2003, Durban, South Africa.

2003 Invited Speaker. Molecular methods for epidemiology of human sleeping sickness. ICPTV, Nairobi, Kenya

2003 Invited talk. *Sodalis glossinidius* tsetse friend or human foe? IV international Congress on symbiosis, International Symbiosis Society, Halifax. Canada.

2003 Speaker. Livestock demography and the risk and the risk of spreading *T. b. rhodesisens* in Uganda – implications for policy. ICPTV, Nairobi, Kenya

2002 Speaker. SRA as a marker for human infectivity in the domestic and wild-animal reservoir in East Africa. 30th Trypanosomiasis and Leishmaniasis Symposium, Edinburgh.

2002 Invited Speaker. Sleeping sickness – human health problem, animal health solution. Infectious Diseases Symposium, The Royal Society, Edinburgh.

2002 Invited Speaker - *Sodalis glossinidius* - Tsetse friend or human foe? VIIth European Congress of Entomology. Thessaloniki, Greece.

1998 Invited Speaker. Death and the single trypanosome, International Colloquium - Sleeping Sickness Rediscovered, Institute of Tropical Medicine, Antwerp, Belgium.

1998 Speaker. Programmed Cell death in procyclic *T. b. rhodesiense.* 29th Trypanosomiasis and Leishmaniasis Symposium, Arachon, France.

1998 Programmed Cell Death in *T. b. rhodesiense*. 1998 2nd International Internet Conference

1997 Invited Speaker. Life and Death of *Trypanosome brucei rhodesiense*. XIII Meeting Brazilian Society of Protozoology, Brazil.

1997 Programmed cell death in *T. b. rhodesiense* is associated with up regulation of mRNAs. Apoptosis and Programmed Cell Death, Keystone Symposia Conferences, U.S.A.

1996 Control of maturation in tsetse. Conference of Entomology, Florence.

1995 Regulation of trypanosome infections in tsetse. British Society of Parasitology, Edinburgh.

1995 Control of infections in tsetse. 28th Trypanosomiasis and Leishmaniasis Seminar, Glasgow

1994 Differential transmission of human infective and non-infective trypanosome stocks from Uganda. XXII ISCTRC Meeting, Kampala, Uganda.

1993 PCR Identification of Rickettsia-like symbionts in tsetse flies, 27th Trypanosomiasis and Leishmaniasis Seminar, London.

1993 Molecular epidemiology of *Trypanosoma brucei* stocks from Uganda: evidence for a multiclonal population structure, 27th Trypanosomiasis and Leishmaniasis Seminar, London

1993 Genetic transformation of non-Drosophilid insects: 'Transformation of insect symbionts', Second International Symposium on Insect Molecular Science, Flagstaff, Arizona, USA.

1992 Inheritance of refractoriness of trypanosome infection in tsetse. Proceedings International Symposium on management of insect pests. IAEA-SM-327/21 195-200. IAEA, Vienna, Austria.

1991 Lectin mediated stimulation of maturation of trypanosome infections in *Glossina*. 20th Meeting of the International Scientific Council for Trypanosomiasis Research and Control, Mombassa, 1989, OAU/STRC, Nairobi, Kenya.

1990 The role of cattle in the epidemiology of sleeping sickness in Uganda. VII International Congress of Parasitology, Paris, France.

1990 Lectins, differentiation and the control of parasite maturation by signals from the tsetse fly. VII International Congress of Parasitology, Paris, France.

Appointments as external examiner for the award of doctoral degree

University of Salford PhD Aimee Tilley 2003 (Examiner); Brunel University PhD Francis Odongo 2004 (Examiner); University of Makerere, Uganda. Charles Waiswa, 2002 (Appointment to Supervisor role); University of Sokoine, Tanzania. Magai Kaare 2004 (Appointment to Supervisor role). Zachara 2006, Makerere University (Appointment to supervisor role)

Articles in Popular Scientific Press & Radio and TV Interviews

- *New Scientist* - Jan 1988 - Sugar pills for tsetse flies.
- *New Scientist* - Feb 1995 - Cell death in parasitic protozoa.
- *Science* - 30 July 1993 - Bacteria may provide access to the tsetse fly.
- *Science Now* - Feb 1996 - Programmed Cell Death in unicellular organisms
- *BBC World Service* - Feb 1996 - Programmed Cell Death in unicellular organisms
- *CNN* - Feb 1996 - Programmed Cell Death in Unicellular organisms
- *New Agriculturalist* - January 2001 - Human sleeping sickness Waking up to reality http://www.new-agri.co.uk/01-1/focuson/focuson4.html
- *BBC Radio 4* - August 2001 – Interview for Parasite series.
- *BBC1 Parasite series* - December 2003 - Featured research on human sleeping sickness.
- Uganda:Sleeping sickness epidemic worries ministry of Health http://medilinkz.org/news/news2.asp?NewsID=2542

- *New Agriculturalist* - November 2005 - Education and training for the African animal health sector http://www.new-agri.co.uk/05-6/pov.html
- *SciDev* – December 2005 - Double parasite threat of sleeping sickness in Uganda http://www.scidev.net/News/index.cfm?fuseaction=readNews&itemid=25078
- *ID21* - December 2005 - Uganda faces double threat from sleeping sickness http://www.id21.org/health/h4kp1g1.html
- *SciDev* - March 2006 - Africa's vets to gain from web-based training http://www.scidev.net/News/index.cfm?fuseaction=readNews&itemid=2726
- *New Agriculturalist* - July 2006 - The need to control sleeping sickness http://www.new-agri.co.uk/06-4/perspect.html
- *Research for Development* – 2006 - Sleeping sickness: waking up to reality? www.research4development.info/caseStudies.asp?ArticleID=189
- Picozzi, K., McOdimba, F., Tilley, A., Fèvre, E., Coleman, P., Magona, J., Odiit, M., Eisler, M.C. and Welburn, S. (2003). Diagnosis of Trypanosoma brucei infections in cattle: applications of novel technologies for estimating disease risk. ICPTV Newsletter, 8, 24-25.
- Magona, J., Anderson, I., Olaho-Mukani, W., Jonsson, N., Revie, C. and Eisler, M.C. (2003). Diagnosis of endemic diseases in village cattle herds in southeast Uganda: a low technology decision support system. ICPTV Newsletter, 8, 43-46.
- Fèvre, E., Magona, J., Coleman, P., Woolhouse, M. and Welburn, S. (2003). Livestock demography and the risk of spreading T. brucei rhodesiense in Uganda: implications for policy. ICPTV Newsletter, 8, 21-22.
- Machila, N., Thuranira, C., Eisler, M.C., Maudlin, I., Shaw, A. and Welburn, S. (2003). Seasonal and socio-economic effects on animal healthcare practices of smallholder farmers in tsetse endemic areas of western and coastal Kenya. ICPTV Newsletter, 8, 47-49.

PUBLICATIONS

a) Books/ book chapters published (7)

Darby A. and S.C. Welburn (2006) Symbiont Culture. In. Insect Symbiosis, Volume II, Eds. Kostas Bourtzis and Tom Miller CRC Press. In press

Welburn, S.C., Picozzi, K., Kaare, M., Fèvre, E.M., Coleman, P.G and T. Mlengeya (2005). Control Options for Human Sleeping Sickness in Relation to the Animal Reservoir of Disease. In Conservation and Development Interventions at the Wildlife/Livestock Interface: Implications for Wildlife, Livestock and Human Health. Eds. Osofsky, S. A., Cleaveland, S., Karesh, W. B., Kock, M. D., Nyhus, P. J., Starr, L., and A. Yang. IUCN, Gland, Switzerland and Cambridge, UK. 220 pp.

Dale, C, Aksoy, S., Welburn, SC ., Maudlin I and A Oren (2005) *Genus* Sodalis *Dale and Maudlin 1999, 267*^{VP} . In Volume 2 Bergey's Manual of Systematic Bacteriology, The Proteobacteria. 2nd Edition. Eds. George M. Garrity, Editor-in-Chief. Springer, New York ISBN 0-387-95040-0

Welburn, S.C., Fevre, EM, Coleman, PG and I Maudlin I (2004). The epidemiology of human sleeping sickness in Uganda. In 'The Trypanosomiases'. Eds. Maudlin, I., Holmes, P and M. Miles. CAB International Oxford.

Welburn, S.C., and I. Maudlin (1996). Current trends in parasite vector interactions (*Leishmania* and *Trypanosoma*). In 'Trypanosomiasis and Leishmaniasis: integrated approaches to control'. Eds. Hide, G., Mottram, J.C., Coombs, G.H. and P.H. Holmes. British Society for Parasitology /CAB International, Oxford. pp. 315-325.

Kaslow, D.C. and S.C. Welburn (1996). Insect-transmitted pathogens in the insect midgut. In 'Biology of the Insect Midgut' CAB International, Oxford (Eds. Lehane, M.J and Billingsley, P). pp. 342-462.

Welburn, S.C and C. Dale. (1997). Isolation and culture of insect symbionts. In 'The molecular biology of insect disease vectors: a methods manual'. (Eds. Crampton, J.M., Beard, C.B. and L. Kitsos). pp547-544.

b) Articles published in peer refereed journals (69)

Welburn, S.C., Picozzi, K., Kaare, M., Cleveland, S., and Mlengela, T. (2007) Identifying reservoirs of infection for human sleeping sickness in the Serengeti ecosystem. *Emerging Infectious Diseases.*

*Kaare, M.T., Picozzi, K., Mlengeya, T., Fèvre, E.M., Mtambo, M.M., Mellau, L.S., Cleaveland, S and Welburn, S.C. (2007) Sleeping sickness – a re-emerging disease in the Serengeti? *Travel Medicine and Infectious Disease. In press*

Fèvre, E.M., Tilley, A., Picozzi, K., Fyfe, J., Magona, J.W., Shaw., D.J., Eisler, M.C., and S.C. Welburn. (2006) Central point sampling from cattle in livestock markets in areas of human sleeping sickness. *Acta Tropica.* 97(2):229-32.

Machila, N., Emongor, R., Shaw, A.P., Welburn, S.C., McDermott, J.J, Maudlin, I and M.C. Eisler (2006). A community education intervention to improve bovine trypanosomiasis knowledge and appropriate use of trypanocidal drugs on smallholder farms in Kenya. *Agricultural Systems.*

Odiit, M., Bessell, P., Fèvre, E.M., Robinson, T., Kinoti, J., Coleman, P.G., Welburn, S.C., McDermott, J.J., and M.E.J. Woolhouse, (2006). Using remote sensing and geographic information systems to identify villages at high risk for *T. b. rhodesiense* sleeping sickness. *Trans R Soc Trop Med Hyg.*

Waiswa, C., Picozzi, K., Katunguka-Rwakishaya, E., Olaho-Mukani, E., and S.C Welburn. (2006) *Glossina fuscipes fuscipes* in the trypanosomiasis endemic areas of south eastern Uganda: apparent density, trypanosome infection rates and host feeding preferences. *Acta Tropica.* E-pub.

Cox, A., Tilley, A., McOdimba, F., Fyfe, J., Eisler, M.C., Hide, G and S.C. Welburn (2005). A PCR based assay for detection and differentiation of African trypanosome species in blood. *Experimental Parasitology* 111: 24-29.

Darby, A.C., Chandler, S.M., Welburn, S.C. and A.E. Douglas (2005). Aphid-Symbiotic bacteria cultured in insect cell lines. *Applied and Environmental Microbiology* 71(8): 4833-9.

*Darby, A.C., Lagnel, J., Matthew, C.Z., Bourtzis, K., Maudlin, I., and S.C. Welburn (2005). Extra-chromosomal DNA of the symbiont *Sodalis glossinidius. Journal of Bacteriology* 187(14): 5003-7.

*Fèvre, E.M., Picozzi, K., Fyfe, J., Waiswa, C., Odiit, M., Coleman, P.G. and S.C. Welburn (2005). A burgeoning epidemic of sleeping sickness in Uganda. *Lancet* 366: 747-747.
Hammarton, T., Lillico, S., Welburn, S.C. and J.C. Mottram (2005) *Trypanosoma brucei* MOB1 is required for accurate and efficient cytokinesis but not for exit from mitosis. *Molecular Microbiology* 56(1): 104-116.

Lambson, B., Nene, V., Obura, M., Shah, T., Pandit, P., Ole-MoiYoi, O., Delroux,K., Welburn, S.C., Skilton, R., de Villiers, E. and R. Bishop (2005). Identification of candidate sialome components expressed in ixodid tick salivary glands using secretion signal complementation in mammalian cells. *Insect Molecular Biology* 14(4): 403-14.

Matthews, C.Z., Darby, A.C., Young, S.A., Hume, L.H. and S.C. Welburn (2005). The rapid isolation and growth dynamics of the tsetse symbiont *Sodalis glossinidius*. *FEMS Microbiology Letters* 248(1): 69-74.

Odiit, M., Fèvre, E., Coleman, PG., Welburn, SC., McDermott, J., and M.E.J Woolhouse. (2005) Quantifying the level of under-reporting of sleeping sickness cases. *Tropical Medicine International Health* 10(9): 840-9.

*Picozzi, K., Fevre, E.M., Odiit, M., Carrington, M., Eisler, M.C., Maudlin, I and S.C. Welburn (2005). Sleeping sickness in Uganda: a thin line between two fatal diseases. *British Medical Journal*. Nov 26;331(7527):1238-41

Fèvre, E.M., Coleman, P.G., Welburn, S.C., and I. Maudlin (2004) Reanalyzing the 1900-1920 sleeping sickness epidemic in Uganda. *Emerging Infectious Diseases* 10(4): 567-73.

Odiit M, Shaw A, Welburn SC, Fevre EM, Coleman PG, and J.J. McDermott (2004) Assessing the patterns of health-seeking behaviour and awareness among sleeping-sickness patients in eastern Uganda. *Annals of Tropical Medicine and Parasitology* 98(4): 339-48.

Odiit, M., Coleman, P.G., McDermott, J.J., Fevre, E.M., Welburn, S.C. and M.E.J. Woolhouse (2004) Spatial and temporal risk factors for the early detection of *Trypanosoma brucei rhodesiense* sleeping sickness patients in Tororo and Busia districts, Uganda. *Transactions of the Royal Society of Tropical Medicine and Hygiene* 98(10): 569-576.

Hutchinson, O. C., Smith, W., Jones, N.G, Charropadhyay, A., Welburn, S.C. and M. Carrington (2003) VSG Structure: similar N-terminal domains can form functi9onal VSGs with different types of C-terminal domain. *Molecular and Biochemical Parasitology* 130(2): 127-31.

Machila, N., Wanyangu, S.W., McDermott, J.J., Welburn, S.C., Maudlin I., and M.C. Eisler (2003) Cattle owners perceptions of African Bovine trypanosomiasis and its control in Busia and Kwale Districts of Kenya. *Acta Tropica* 86(1): 25-34.

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Curriculum Vitae

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EDUCATION:

- Doctor of Philosophy (PhD) in Veterinary Parasitology from Royal Veterinary & Agricultural University, Denmark (1996)
- Doctor of Veterinary Medicine (DVM) from University of Georgia, USA (1986)
- Bachelor of Science (BS) in Zoology and Psychology from University of Georgia, USA (1981)

DISSERTATION:

- "Experimental *Schistosoma japonicum* infection in the pig: the host regulatory response and other aspects of the host-parasite relationship"

LICENSURE:

- Licensed since 1986 to practice Veterinary Medicine, State of Georgia, USA

AREAS OF SPECIALIZATION:

- Parasitology
- Parasitic Zoonoses of both agricultural and public health importance in developing countries (*e.g.* cysticercosis, zoonotic schistosomiasis, echinococcosis)
- Veterinary Public Health
- Development Assistance

WORK EXPERIENCE:

- Scientific Coordinator of DANIDA-funded Research Capacity Strengthening project "Cross-Disciplinary Risk Assessment of Cysticercosis in Eastern and Southern Africa" based at Danish Center of Experimental Parasitology, Royal Veterinary and Agricultural University, Denmark (2006 – present)

- Danida secondment to the International Livestock Research Institute Headquarters in Nairobi, Kenya to work with the "People, Livestock and the Environment" thematic area advising how to include research and control activities on parasitic zoonoses of agricultural and public health importance in the PLE thematic programme. Cysticercosis is the primary focus due to its strong linkage with poverty (2004 – 2006).

- External Associate Professor, Royal Veterinary and Agricultural Univeristy, Frederiksberg, Denmark (2004 – present).

- Project leader for Danida-funded Research Collaboration Programme between Mozambique and Denmark "Improving Smallholder Pig Production and Health Project" being responsible as the lead partner from Denmark (2004 – 2005).

- Project leader for U. S. National Institutes of Health-funded project on "Ecology and Transmission of schistosomiasis japonica in the Philippines" being responsible for animal parasitology aspects (2001 – present).

- Coordinator, Danida-funded Livestock Helminths Research Project in Eastern and Southern Africa aimed at enhancing research capacity in veterinary helminthology at veterinary faculties in Kenya, Tanzania, Zambia and Zimbabwe by supporting masters and doctoral students conducting research on important helminth diseases of livestock belonging to rural smallholder farmers (1997 – 2004).

- Senior Research Scientist, Department of Veterinary Microbiology, Royal Veterinary & Agricultural University, Frederiksberg, Denmark (1997 – 2004)

- Fulbright Research Fellow, Laboratory for Parasitology, Royal Veterinary & Agricultural University, Frederiksberg, Denmark for conducting experimental research on schistosomiasis japonica in pigs (January – November, 1993).

- U. S. Peace Corps Volunteer Veterinarian in Boulemane Province, Morocco providing veterinary services to rural sheep and goat farmers and assisting health and agriculture ministries with securing resources for cystic echinococcosis research and control. (1990 – 1992).

- Relief Veterinarian, animal hospitals in Atlanta, Forest Park and Macon, Georgia, USA (1989 – 1990).

- Associate Veterinarian, Wesleyan Animal Hospital, Macon, Georgia, USA (1986 – 1989).

PUBLICATION/PAPERS:

- Sikasunge C.S., Phiri I.K., Phiri A.M., Dorny P., Siziya S., Willingham III A.L. (2006). Risk factors associated with porcine cysticercosis in selected districts of Eastern and Southern Provinces of Zambia. *Veterinary Parasitology* (in press).

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GRANTS:

- Awarded Conference venue at Rockefeller Foundation's International Conference Center in Bellagio, Italy for 3 day conference on "Implementing a Global Campaign for Combating Cysticercosis" which includes use of the conference facilities, meals, accommodation and international travel for participants coming from developing countries (September, 2006).
- $1.4 million from DANIDA for "Cross-Disciplinary Risk Assessment of *Taenia solium* Cysticercosis in Eastern and Southern Africa" (2006-2009).
- Awarded Conference venue at Rockefeller Foundation's International Conference Center in Bellagio, Italy for 3 day conference on "Establishing a Global Campaign for Combating Cysticercosis" which included use of the conference facilities, meals, accommodation and international travel for participants coming from developing countries (September, 2004).
- $50,000 from the Izumi Foundation for "Establishing a Surveillance and Diagnosis Framework for Cysticercosis in Eastern and Southern Africa" (2004-2005).
- $130,000 from Danida supported Agricultural Research fund in Mozambique for the project "Improving Smallholder Pig Production and Health" (2004 – 2005).
- $30,000 from Danida for formulating a project proposal on "Improving Smallholder Pig Production and Health in Eastern and Southern Africa" (2003 – 2004).
- $15,000 from WHO Headquarters for "Assessing the Global Burden and Impact of *Taenia solium* Cysticercosis" (2003-2004).
- $200,000 from Danida-funded Institutes and Projects, World Bank, WHO, FAO, Wellcome Trust, National Research Foundation of South Africa, Stiftelsen Adiutor Foundation, and Virbac Pharmaceutical Company for "International Action Planning Workshop on *Taenia solium* Cysticercosis/Taeniosis with Special Focus on Eastern and Southern Africa" held in Arusha, Tanzania from 19 – 22 August, 2002.
- $1.1 million from DANIDA for ENRECA Livestock Helminths Research Project in Eastern & Southern Africa (4th Phase, 2001-2004).
- $1.6 million from U. S. National Institutes of Health for "Ecology and Transmission of *Schistosoma japonicum* in the Philippines" Project (2001-2006).

HONORS/AWARDS/DISTINCTIONS

- Invited Speaker and Session Chair, 5th European Congress on Tropical Medicine and International Health, Amsterdam, Netherlands, May, 2007.

- Invited Participant, International Conference on Neglected Infectious Diseases "How to meet the challenge for Europe's international research cooperation in the field of Neglected Infectious Diseases" in Brussels, Belgium, November, 2006

- Invited Consultant to the "WHO Consultation on Formulating Strategy for Assessing the Burden of Food Borned Diseases" in Geneva, Switzerland, September, 2006.

- Invited Expert to the "WHO/DFID-AHP Meeting on the Control of Zoonotic Diseases: A Route to Poverty Alleviation" at WHO Headquarters in Geneva, Switzerland, September, 2005.

- Photograph "Female farmer in Mbulu District, Tanzania with pigs" placed on cover of international journal *Trends in Parasitology*, July 2005 issue.

- Invited Consultant to the "WHO/FAO/OIE Expert and Technical Consultations on Strengthening Regional Capacity for Surveillance and Control of Zoonoses" in Rome, Italy, June, 2005.

- Invited Expert to the "WHO/FAO/OIE Consultation on Emerging Zoonoses" in Geneva, Switzerland, May, 2004. Appointed Rapporteur for the Consultation.

- Invited symposium organizer ("Livestock Helminthoses of Public Health Significance") for the International Congress of Zoology in Beijing, China, 23 – 27 August, 2004.

- Invited Scientific Committee Member and Symposium co-organizer ("Cysticercosis in Asia and Africa") for the Food-Borne Parasitic Zoonoses meeting in Bangkok, Thailand, December, 2003.

- Invited Expert to the "FAO Expert Consultation on Community-based Veterinary Public Health" in Rome, Italy, October, 2003. Appointed Secretary for the Expert Consultation.

- Invited symposium organizer for symposium on "Assessing the Burden of *Taenia solium* Cysticercosis and Echinococcosis" at the Conference of the World Association for the Advancement of Veterinary Parasitology, New Orleans, Louisiana, USA, August, 2003.

- Invited speaker to the International Task Force for Disease Eradication Meeting to speak on the status of *Taenia solium* cysticercosis, Carter Center, Atlanta, Georgia, USA, 16 April, 2003.

- Invited speaker to the Centenary Symposium to Celebrate the Discovery of *Schistosoma japonicum*, Kurume, Japan, 31 March, 2003.

- Appointed advisor for WHO-funded initiative on the assessment of the burden and impact of *Taenia solium* cysticercosis around the world, 2003 – 2004.

- Invited to serve as section editor for WHO/FAO/OIE Manual on Taeniosis and Cysticercosis in Man and Animals: Detection, Treatment and Prevention to be published in 2003.

- Invited Keynote Speaker to European Tropical Medicine to speak on "Initiation of a Regional Programme for Surveillance, Prevention and Control of Taenia solium Cysticercosis/Taeniosis in Eastern & Southern Africa", Lisbon, Portugal, 9 – 12 September, 2002

- Chief organizer "International Action Planning Workshop on *Taenia solium* Cysticercosis/Taeniosis with Special Focus on Eastern & Southern Africa" held in Arusha, Tanzania from 19 – 22 August, 2002

- Serving as coordinator for "Research on Animal Reservoir Hosts" component for Strengthening Regional Network of Asian Schistosomiasis project funded by WHO/TDR

- Invited speaker for Workshop on "Cysticercosis", International Tropical Medicine Congress, Lisbon, Portugal, September, 2002

- Invited speaker for Symposium on "Water & Food-borne Parasitic Zoonoses", International Congress of Parasitology (ICOPA), August, 2002
- Invited as consultant for Danida-funded Wastewater Reuse Project in Vietnam to assist Vietnamese scientists formulate a review of cysticercosis in Vietnam, January, 2002

- Served as Organizer and Moderator for WHO/FAO/UNEP Panel of Experts on Environmental Management of Vectors workshop on "Environmental modification and livestock management for control of zoonotic schistosomiasis in the Philippines", November, 2001.

- Invited as an author for DIFID-funded document on "Livestock Health Research Opportunities for Poverty Alleviation" for the section devoted to meat-borne and other parasitic zoonoses.

- Appointed as Deputy Director of new WHO/FAO Collaborating Centre for Research and Training on Emerging and Other Parasitic Zoonoses, April, 2001.

- Invited to attend National Experts meeting in China on reviewing and evaluating a draft plan for a new national survey on the current status of important parasitoses (other than malaria, schistosomiasis and filariasis) with emphasis on geohelminths and parasitic zoonoses organized by the Chinese Ministry of Health, Beijing, People's Republic of China, 2001.

- Awarded a grant from the Department of Microbiology, Royal Veterinary & Agricultural University to participate in the 3rd Conference on Food-Borne Parasitic Zoonoses held in Bangkok, Thailand and then go to Kathmandu, Nepal to do a situation analysis with regard to porcine cysticercosis and assist in formulating a proposal for epidemiological studies on the zoonosis in a rural smallholder farming community where pig keeping is popular, Kathmandu, Nepal, 2000.

- Invited to attend an experts meeting on parasitic zoonoses in underdeveloped western China, Chengdu, Sichuan Province, People's Republic of China, 2000.

- Invited as a Visiting Professor to the Sichuan Institute of Parasitic Diseases, Chengdu, Sichuan Province, People's Republic of China, 1999.

- Appointed to the Cysticercosis Working Group of the European Commission's Scientific Committee on Veterinary Measures relating to Public Health, 1999.

- Invited to serve as a Session Chairman at the Scandinavian Symposium for Parasitology, Reykjavik, Iceland, 1999.

- Invited to serve as Co-Chairman of a session on parasitic zoonoses, 17th International Conference of the World Association for the Advancement of Veterinary Parasitology, Copenhagen, Denmark, 1999.

- Invited to participate as a veterinary consultant at the World Health Organization's Informal Consultation on Schistosomiasis Control at WHO Headquarters, Geneva, Switzerland, 1998.

- Appointed veterinary advisor to the People's Republic of China/Republic of the Philippines Regional Networking Group on Schistosomiasis, 1998.

- Selected as an alternate candidate for U. S. Centers for Disease Control and Prevention *Epidemic Intelligence Service* (EIS) Program, 1997.

- Awarded AAVP Outstanding Graduate Student Award by the American Association of Veterinary Parasitologists, 1996.

- Awarded Samuel F. Scheidy Memorial Award by the American Veterinary Medical Foundation for Excellence in Quality of Veterinary Medical Research Reported at the World Veterinary Congress, 1995.

LANGUAGES:

- English: mother tongue
- Danish: Read – good; Write – fair; Speak - fair
- Spanish: Read – fair; Write – slight; Speak - slight
- French: Read – fair, Write – slight: Speak - slight
- Arabic (Moroccan dialect): Read - slight; Write - slight; Speak - slight

PROFESSIONAL AFFILIATIONS /MEMBERSHIPS:

- American Veterinary Medical Association
- Omega Tau Sigma Veterinary Fraternity
- Danish Society of Parasitologists
- Scandinavian Society of Parasitology
- American Society of Parasitologists
- American Association of Veterinary Parasitologists
- American Society of Tropical Medicine & Hygiene
- World Association for Advancement of Veterinary Parasitology
- Advisor, Regional Network for Asian Schistosomiasis
- Advisor, Cysticercosis Working Group in Eastern and Southern Africa
- Friends of Morocco
- Association for Returned Peace Corps Volunteers

REFERENCES

Prof. Stig Thamsborg, Director Danish Centre for Experimental Parasitology & Head WHO/FAO Collaborating Centre for Research & Training on Parasitic Zoonoses, Royal Veterinary & Agricultural University, Frederiksberg, Denmark (tel: +45 35282775; fax: +45 35282774; e-mail: smt@kvl.dk)

Dr. Peter Schantz, Veterinary Director Parasitic Diseases Division, National Center for Infectious Diseases, U. S. Centers for Disease Control & Prevention, 4770 Buford Hwy NE, Atlanta, Georgia, USA 30341 (tel: +1 770 488 7767; fax: +1 770 488 7761; e-mail: pms1@cdc.gov)

Dr. Niels Ørnbjerg Christensen, Director, DBL – Institute for Health Research and Development, Jaegersborg Alle 1 D, 2920 Charlottenlund, Denmark (tel: +45 77337733; fax: +45 77337732; e-mail: nornbjerg@dblnet.dk)

Dr. Lorenzo Savioli, Director, Department of Control of Neglected Tropical Diseases (NTD), Division of Communicable Diseases Control, Prevention and Eradication, World Health Organization, 20 Avenue Appia, 1211 Geneva 27, Switzerland (tel: +41 22 79 12664/14729; fax: +41 22 79 14869; e-mail: saviolil@who.int)

Dr. Carlos Eddi, Senior Officer Parasitology, Animal Health Service, Animal Production and Health Division, Food and Agriculture Organization, Room C-528, Viale delle Terme di Caracalla, 00100 Rome, Italy (tel: +39 0657054159; fax: +39 0657055749; e-mail: carlos.eddi@fao.org)

Prof. Erastus Olonde Amayo

Curriculum vitae as at October 2006

PERSONAL DATA

Name: Prof. Erastus Olonde Amayo M.B, Ch. B,M. Med (Internal Medicine)

Certificate in Medical Anthropology

Nationality: Kenyan

Date of Birth: 14th November 1957

Age: 48 years

Marital Status ; Married with 3 children

Qualifications

Mb.ChB, M. Med(Internal Medicine), Certificate in Tropical Medicine, Certificate in Health and Behavior Research (Harvard)

UNIVERSITY EDUCATION

1999 March- May Harvard Medical School Fellowship in Medical Anthropology, Department of Social Medicine USA

1996-1997 Harvard Medical School Fellowship in Medical Anthropology ,

1992-1993 University of Edinburgh Fellowship in Clinical Neurology at the, Western General Hospital Scotland U.K

1985-1988 University of Nairobi Masters in Medicine (Internal Medicine)

1977- 1983 University of Nairobi, qualified with a Mb, Ch.B degree

ACADEMIC CERTIFICATES

1997 Certificate In Health and Behavior Research, Harvard Medical School U.S.A

1988: Masters of Medicine(Internal Medicine) University of Nairobi

1983: Bachelor of Medicine , Bachelor of Surgery . University of Nairobi

1976: East African Advanced Certificate of Education "A" levels passed with 3 principles and one subsidiary

1974: East African Certificate of Education"O" level certificate with 1st Division

1970: Certificate of Primary Education Passed

CURRENT APPOINTMENT

Associate Professor - Department of Clinical Medicine and Therapeutics

Honorary Consultant Physician and Neurologist Kenyatta National Hospital

Thematic head Clinical Medicine

PREVIOUS POSITIONS

1997-2004 Senior Lecturer , Department of Medicine University of Nairobi
1988-1997 Lecturer , Department of Medicine University Of Nairobi

1986-1988 Tutorial Fellow Department of Medicine University of Nairobi

1985-1986 Senior House Officer Kenyatta National Hospital

1984-1985 Medical Officer Nanyuki District Hospital

1983-1984 Medical Officer Intern Machakos hospital

PROFESSIONAL TRAINING

1996-1997 Clinical Electroencephalography and EMG Brigham and Women Hospital Boston U. S. A

May 1992 – May 1993 Training in Clinical Neurology Western General Hospital Edinburgh Scotland

OUTSTANDING PERFORMANCE AND AWARDS

1) M.B.Ch.B Distinction in Biostatistics ,Credit passes in Medicine, Physiology, Biochemistry, General Pathology and Human Anatomy.

2) Certificate in Tropical Medicine Credit pass
3) Wellcome Trust Prize for the best M. Med dissertation in Medicine in 1998.
4) Commonwealth Medical Association fellowship to study clinical Neurology
5) Carnegie Co-operation Fellowship to study social Medicine at Harvard Medical School USA
6) Carnegie Co-operation Fellowship to study Social Medicine at Harvard Medical School U.S.A

SPECIALISED SKILLS

EMG

EEG

Research methodology

computer

MEMBERSHIP OF SOCIETIES AND PROFESSIONAL BODIES

1) American Academy of Neurology
2) American Heart Association (Stroke council Member)
3) Kenyan Association of Physicians
4) Kenya Medical Association
5) Kenya Society of Epilepsy

ACADEMIC PUBLICATIONS

Amayo E. O. Neurological letter from Kenya
Practical neurology 2006 ;6;261-262

Lwai-Lume L, Ogutu E. O. **Amayo E. O,** Kariuki S. Drug susceptibility pattern of helicobacter Pylori in patients with dyspepsia at the Kenyatta National Hospital Nairobi East African Medical Journal 2005Vol 82;12,603-608

Hooker JA, Muhindi DW, **Amayo EO**, Mc'ligeyo SO, Bhatt KM, Odhiambo JA
Diagnostic utility of cerebrospinal fluid studies in patients with clinically suspected tuberculous meningitis.
Int J Tuberc Lung Dis. **2003** Aug;7(8):787-96.

Nyamu PN, Otieno CF, **Amayo E O**, McLigeyo S O
Risk factors and prevalence of Diabetic foot ulcers at Kenyatta National Hospital , Nairobi East African Medical Journal **2002** Vol. 80 1,36-43

Amayo E. O, Jowi J. O, Njeru E.K
Headache associated disability in Medical students at the Kenyatta National Hospital East African Medical Journal **2002** Vol. 79,10,519-523

Amayo E. O, Kayima J. K, Joshi M. D
Platelet functions in patients with meningococcal meningitis at the Kenyatta National Hospital, Nairobi
East African Medical Journal **2002** Vol.79:8: 405-407

Wanjohi F. W, Otieno F. C. F, Ogola E. N, **Amayo E. O**
Nephropathy in patients with recently diagnosed type 2 diabetes mellitus in Black African
East African Medical Journal **2002** Vol. 79:8: 399-404

Amayo E. O, Kwasa T. O, Otieno C. F
Herpes zoster myelitis: report of two cases
East African Medical Journal **2002**: Vol.79,5,;279-280

Lule G. N, **Amayo E.O**
Irritable Bowel syndrome in Black Kenyans
East African Medical Journal **2002** Vol 79,7,360-363

Amayo E. O, Kwasa T. O, Musau C. K, Mugo G, Wambani. J
Primary intracerebral hemorrhage complicated by cerebral abscess: Case report and review of literature
East African Medical Journal **2002**.Vol 79,3;163-164

Karari E. M, Lule G. N, Mcligeyo S. O, **Amayo E. O**.
Endoscopic findings and the prevalence of Helicobacter pylori in chronic renal failure patients with dyspepsia.
East African Medical Journal **2000**: Vol 77. 406-409.

Mary-Jo DelVecchio Good, **Amayo E O**, Machoki J. M, Mwaikambo E.
Clinical realities and moral dilemmas. Contrasting perspectives from Academic medicine in Kenya, Tanzania and America
Journal Of the American Academy of Arts and Sciences **2000**: Vol 128,6,167-196.

Amayo E. O, Kayima J. K, Amayo A. A.
Transient Focal neurological deficits in patients with hypoglycemia and hyperglycemia; report of 4 cases
East African Medical Journal **1998**:Vol.75;53-54

Amayo E. O, Jowi J. O, Njeru E. K.
Migraine headaches in a group of medical students at the Kenyatta National Hospital Nairobi
East African Medical Journal 1996: Vol 73;594-597

Mbuya S. O, Kwasa T. O, **Amayo E. O**, Kioy P. G, Bhatt S. M.
Peripheral Neuropathy in AIDS patients at Kenyatta National Hospital
East African Medical Journal 1996:Vol 73;538-539

Mcligeyo S. O, Mbui J, Kungu A, **Amayo E O**, Ogendo S. W. O.
Fibrosarcoma of the lung with extrapulmonary manifestation; A scase report
East African Medical Journal 1995: Vol 72;465

Kwasa T O, Jowi J O, **Amayo EO**
Efficacy and tolerability of oral sumatriptan in the treatment of acute migraine
East African Medical Journal 1995. Vol 72;479

Lindley RI , **Amayo E O**, Marshall J, Sandercock P AG, Dennis M, Warlow CP
Acute stroke treatment in UK Hospitals; The stroke Association Survey of Consultant Opinion
Journal Of the Royal College Of Physicians of London 1995:29 no 6 479-484

Lindley RI , **Amayo E O**, Marshall J, Sandercock P AG, Dennis M, Warlow CP
Hospital services for patients with acute stoke in the United Kingdom: The stroke Association Survey of Consultants Opinion
Age and aging 1995: 24;525-532

Amayo E O, Kayima J, Kioy P, Mcligeyo S
Autonomic dysfunction in patients with chronic renal failure
East African Medical Journal 1994: 71;253-255

Amayo E O, Riyat M S, Okelo GBA, Adam MA, Toroitich K
Disseminated histoplasmosis in a patient with AIDS . A case report
East African Medical Journal 1993.Vol 70,93-94.

Amayo E O, KwasaT O.
HIV and acute peripheral facial nerve palsy
East African Medical Journal 1991:68:948

Amayo E O, Owade J N, Aluoch J R, Njeru E K
Neurological complications of Sickle Cell Anaemia at the Kenyatta National Hospital : A 5 year retrospective study
East African Medical Journal 1992: 69,660

Ogutu E, **Amayo E. O**, Okoth F G, Lule G N
The prevalence of Hepatitis B surface antigen(HbsAg) . anti-hepatitis B surface (anti-Hbs) and anti-hepatitis b core (anti-Hbc) in patients with AIDS.
East African Medical Journal 1990; 67,355

Amayo E. O
Some aspects of AIDS at Kenyatta National Hospital
Medicus 1988 Vol. 7 No.7

Amayo.E. O
Clinical manifestation of AIDS at the Kenyatta National Hospital
M. Med(Internal Medicine) Dissertation 1988

CONFERENCES ATTENDED AND PAPERS PRESENTED

1) 58th Annual Meeting of the American Academy of Neurology San Diego California April 1-8th 2006

2) World congress of Neurology Sydney Australia 5-11 November 2005: **Impact of backache on medical workers at the Kenyatta National Hospital**

3) 57th Annual Meeting of the American Academy of Neurology
Miami Beach Florida April 9th-16 2005

4) Cysticercosis working group of Eastern and South Africa
11-13th November 2004 Maputo Mozambique

5) 56th Annual Meeting of The American Academy Of Neurology
April 24th –May 1st 2004 San Francisco California U. S.A

6) 13th ICASA conference 21st to 26th September 2003 Nairobi Kenya

7) Neurological society of Kenya April 2003.What is the evidence for surgical evacuation of intracerebral heamorrhage?

8). Kenya Association of Physicians
Seventh annual Scientific conference 26th to March 2003 Nairobi

Papers presented

1. Is it possible to have ethical practice in resource poor setting?

2. Prevention of stroke beyond Aspirin.

3. Health and social change in East Africa April 14-15 1999 Boston USA. Paper presented.Medical practice in the era of scarcity and HIV/ AIDS

4. World Federation of Public Health Association Conference 12-17th October 1997, Arusha Tanzania.

5. 5.th Inter-Faculty Collaborative Program annual conference 17-22nd August 1997 Matuu. Paper presented; Adherence in therapy in chronic disease.

6. Health and social Change in East Africa. Paper presented; socio-cultural aspects of Epilepsy; implication on treatment adherence 25th April 1997 Boston USA

7. 49th Annual Meeting of the American Academy of Neurology 12-19th April 1997 Boston USA

8. 14th International Conference on the Social Sciences and Medicine. 2-6th September 1996. Peebles Scotland UK

9. Inter-Faculty collaborative Program annual meeting August 1996. Machakos. Paper presented: Impact of headache on Medical students at KNH

10. 2nd Conference of Medical Associations and Societies of Kenya. 27th –29th September Nairobi Kenya. Paper presented; Platelet function in patients with meningococal meningitis at KNH.

11. 2nd International Stroke Conference 12-15 May 1993 Geneva, Switzerland. Paper presented: Survey into the care of Acute Stroke by Physicians in the United Kingdom.

12. British Brain Research Group Meeting March 1993 Birmingham U.K

13. Epilepsy Europe 1-5th September 1992, Glasgow Scotland UK

14. 36th Association of Physicians of East Africa Conference 2nd- 5th September 1991, Nairobi, Kenya.

15. 35th Association of Physicians of East Africa Conference. 2nd –5th September 1989, Arusha Tanzania

16. 3rd International Conference on AIDS and associated cancers in Africa , Arusha Tanzania ,14th-16th September 1988.Paper presented; Neurological; manifestation of AIDS at the Kenyatta National Hospital

COURSES ATTENDED

1. CWGESA Cycticercosis/Taeniosis awareness and training workshop University of Transkei Umtata Eastern Cape Province , South Africa 7th to 11th June 2004.
2. Leadership and management program workshop 21 May 2004 Nairobi
3. HIV update: Cotemporary Issues in management Boston USA 2-4th May 1999.
4. Grant writing workshop 30th March 1997 Boston USA
5. 15th advanced Clinical Neurology course 31st March to April 1993 Edinburgh Scotland
6. Epidemiology for Clinician Course 4th-8th January 1993 Southampton United Kingdom
7. Stroke course, University of Edinburgh 9th-13th November 1992.
8. Elementary data analysis course at the University Of Edinburgh October, November, December 1992.
9. SPSS course University of Edinburgh Scotland UK.
10. dbase 3 plus at the University of Edinburgh 3rd October 1992

CURRENT RESEARCH

Low backache among health workers at Kenyatta National Hospital.

EEG abnormalities in Patient with sickle cell Anemia

Adherence of therapy to antiretroviral

Peripheral neuropathy in HIV/AIDS

Neurcysticercosis in Kenya

RESEARCH INTERESTS

Cerebrovascular diseases

Neuroepidemiology

Drug adherence

NATIONAL DUTIES

1) Member of the Committee for Drug Registration of the Pharmacy and Poisons Board Ministry of Health.

ADMINISTRATIVE EXPERIENCE

Secretary Association of Physicians of East and Central Africa 1990 –1992

Member of the steering Committee of the Inter-Faculty collaborative program

Secretary Kenya Society of Epilepsy.

Appendix II: CWGESA Questionnaire

TAENIOSIS/CYSTICERCOSIS QUESTIONNAIRE

Last name : _____ First Name : _____

If Child then : Questionnaire number : _____

Father's Name :_____ District _____

Division: _____

Mother's Name:_____ Location: _____

Sub-location: _____

Village _____

Hut (house) number _____

How long have you lived in this village?____ (yrs.)

GPS Reading North:_____(Format N00.xxxxx)

East:: _____(Format E00.xxxxx)

Altitude: _____(Format xxxx m)

1 How old are you? _____ (years)

2 What is your date of birth? ____ Day _____ Month _____ Year

3 Sex ☐ Male ☐ Female

4 What is the highest schooling grade you have completed?

☐ None ☐ Primary school

☐ Middle School ☐ High school

5 What further education have you completed?

☐ None ☐ College

☐ University ☐ Technical/Vocational

6 What is your occupation? _____

7 How many days of work have you missed because of illness in the past month? _____ days

8 How many days of work have you missed because of illness in the past year? _____ days

9 Where do you usually get your drinking water?

☐ River ☐ Bore-hole

☐ Well ☐ Other (please specify) _____

10 Do you boil your drinking water?

☐ Always ☐ Almost always

☐ Sometimes ☐ Never

11 How often do you eat pork?

☐ At least once a month ☐ Less than once a month but at least once a year

☐ Less than once a year ☐ Never

12 How is the pork that you eat prepared? *Check all that apply.*]

☐ Boiling ☐ Barbeque

☐ Fried ☐ Others (specify)_____

13 Do you have a latrine at home?

☐ Yes ☐ No (Skip to Q 14)

13.1 How often do you use a latrine when you have to defecate?

☐ Always ☐ Sometimes ☐ Never

14 Do you keep pigs?

☐ Yes ☐ No (Skip to Q 15)

14.1 What type of pigs do you keep?

☐ Foreign ☐ Native

☐ Both foreign and native ☐ Can not remember, do not know

14.2 Of the pigs that you have, how many are for? [*read each choice and record the number*]

Home consumption _____ Trading _____

Reproduction _____ Other (specify): _____

14.3 How do you keep your pigs… [*read questions 14.3.1 to 14.3.4 one after the other*]

14.3.1 During the planting season

☐ In a pen ☐ Free ranged

☐ Tethering ☐ Other (specify): _____

14.3.2 During the growing season

☐ In a pen ☐ Free ranged

☐ Tethering ☐ Other (specify): _____

14.3.3 During the harvesting season

☐ In a pen ☐ Free ranged

☐ Tethering ☐ Other (specify): _____

14.3.4 During the fallowing season

☐ In a pen ☐ Free ranged

☐ Tethering ☐ Other (specify): _____

14.4 What do your pigs eat? [*Check all that apply.*]

☐ Pasture ☐ Kitchen left overs

☐ Commercial feeds ☐ Other (specify): _____

14.5 How often do you slaughter pigs at home?

☐ At least once a month ☐ Less than once a month but at least once a year

☐ Less than once a year

☐ Never (Skip to Q 14.6) ☐ Can not remember, do not know (Skip to Q 14.6)

14.5.1 If ever, how often was the meat inspected by a meat inspector?

☐ Always ☐ Almost always

☐ Sometimes ☐ Never

☐ Can not remember, do not know

14.6 . What price do you usually sell your pigs when they are ready to be slaughtered (specify the currency used, this can be money or barter)? _____

_____

14.7. What price do you usually sell your piglets (aged 4 months or less) (specify the currency used, this can be money or barter)? _____

_____ (SKIP TO Q 16)

15. Have you ever owned pigs? *[If they answer "yes", ask when they owned pigs]*

☐ Yes, in the past year ☐ Yes, one (1) to five (5) years ago

☐ Yes, more than five (5) years ago

☐ No (skip to Q 17)

15.1. What kind of pigs were they?

☐ Foreign ☐ Native

☐ Both foreign and native ☐ Can not remember, do not know

16. Were you ever told that your pigs or piglets were infected with cysts (cysticercosis)?

☐ Yes ☐ No (Skip to Q 17)

16.1. When were you told that your pig or piglets were infected with cysts (cysticercosis)?

☐ In the past year ☐ One (1) to five (5) years ago

☐ More than five (5) years ago

☐ Never told (skip to Q 17) ☐ Can not remember, do not know (skip to Q 17)

16.1.1 When that happened, were you able to sell your pig(s) or piglets?

☐ Sold both ☐ Sold pigs but not piglets

☐ Sold piglets but not pigs (skip to Q 16.1.3)

☐ Could not sell either (skip to Q 17)

☐ Can not remember, do not know (skip to Q 17)

16.1.2 When that happened, what price did you sell your pigs (aged more than 4 months) (specify the currency used, this can be money or barter)? _____

_____

16.1.3 When that happened, what price did you sell your piglets (aged 4 months or less) (specify the currency used, this can be money or barter)? _____

_____

17. Have you ever seen or heard of white nodules (rice) in pig carcasses?

☐ Yes ☐ No (Skip to 18)

17.1 Where can you find nodules on a live pig?

☐ It is not possible to find them on a live pig

☐ Under the skin ☐ Under the tongue

☐ I don't know ☐ Somewhere else (Specify) _____

17.2 How do pigs get these nodules?

☐ By eating human faeces ☐ By eating pig faeces

☐ From another infected pig ☐ Other (Specify) _____

☐ I don't know

17.3 What would you do if you discovered that your pig had nodules?

☐ Sell the pig ☐ Treat it with herbs

☐ Pierce the nodules ☐ Other (Specify) _____

☐ I don't know

18. Have you ever heard of tapeworm infection in humans?

☐ Yes ☐ No (Skip to question 19)

18.1 How did you learn about it?

☐ By a doctor ☐ By a friend or family member

☐ By a traditional healer ☐ On the radio / newspaper

☐ Other (Specify) _____

18.2 How does a person know if they have a tapeworm?

☐ They can see it in their faeces ☐ They have diarrhea

☐ They have fever ☐ Other (Specify) _____

☐ I don't know

18.3 Have you ever had a tapeworm or seen small parts (segments) of worms in your faeces? (*Show photographs of proglottids*)

☐ Yes ☐ No (SKIP TO Q 18.4)

☐ I don't know/can not remember (SKIP TO Q 18.4)

18.3.1 When that happened, what did you do? [*check all that applies*]

☐ Went to a primary health care provider (hospital, clinic, dispensary)

☐ Went to the pharmacy to get a drug to treat it

☐ Went to a traditional healer ☐ Did nothing

☐ I can not remember, I do not know

18.4 How does a person get tapeworm infection?

☐ They do not wash their hands ☐ They eat undercooked pig meat

☐ They are in contact with an infected person ☐ Other (Specify) _____

☐ I don't know

19. Have you ever had skin nodules or hard lumps under the skin? (*Show photograph of person with subcutaneous cysticercosis nodules*)

☐ Yes, currently has ☐ Yes in the past year, but not currently

☐ Yes, one year or more ago, but not currently ☐ No

☐ Can not remember, do not know

20. Have you ever had bad headaches that lasted more than a few days?

☐ Yes, currently has ☐ Yes in the past year, but not currently

☐ Yes, one year or more ago, but not currently ☐ No

☐ Can not remember, do not know

21. Have you ever had any of the following?

21.1 Sudden loss of consciousness and episodes of incontinence or foaming of the mouth or tongue biting?

☐ Yes, currently has ☐ Yes in the past year, but not currently

☐ Yes, one year or more ago, but not currently

☐ No (Skip to Q 21.2) ☐ Can not remember, do not know (Skip to Q 21.2)

21.1.1 (If yes) How often has this happened?

☐ Only once ☐ More than once

21.1.2 How old were you when this first happened? _____ years

21.2 A brief period of absence(s) or loss(es) of contact with the surroundings that starts suddenly?

☐ Yes, currently has ☐ Yes in the past year, but not currently

☐ Yes, one year or more ago, but not currently

☐ No (Skip to Q 21.3) ☐ Can not remember, do not know (Skip to Q 21.3)

21.2.1 How often has this happened?

☐ Only once ☐ More than once

21.2.2 How old were you when this first happened? _____ years

21.3 Uncontrollable twitching or jerking or abnormal movements of one or more limb(s) (convulsions) that starts suddenly and lasts for a period of a few minutes?

☐ Yes, currently has ☐ Yes in the past year, but not currently

☐ Yes, one year or more ago, but not currently

☐ No (Skip to Q 21.4) ☐ Can not remember, do not know (Skip to Q 21.4)

21.3.1 How often has this happened?

☐ Only once ☐ More than once

21.3.2 How old were you when this first happened? _____ years

21.4 Sudden onset of a brief period of hearing or smelling or seeing things that are not there or feeling strange body sensations?

☐ Yes, currently has ☐ Yes in the past year, but not currently

☐ Yes, one year or more ago, but not currently

☐ No (Skip to Q 21.5) ☐ Can not remember, do not know (Skip to Q 21.5)

21.4.1 How often has this happened?

☐ Only once ☐ More than once

21.4.2 How old were you when this first happened? _____ years

21.5 Were you ever told that you had epilepsy or that you had had an epileptic seizure?

☐ Yes, currently has ☐ Yes in the past year, but not currently

☐ Yes, one year or more ago, but not currently ☐ No

☐ Can not remember, do not know

21.6 Have you ever had seizures or fits?

☐ Yes, currently has ☐ Yes in the past year, but not currently

☐ Yes, one year or more ago, but not currently

☐ No (skip to Q 22) ☐ Can not remember, do not know (skip to Q 22)

21.6.1 How often has this happened?

☐ Only once ☐ More than once

21.6.2 How old were you when this first happened? _____ years

[If the interviewee has answered "no" to questions 21.1-21.6, the interview is finished. Go to last page and complete questions 30 & 31 based on observation.]

THANK YOU VERY MUCH FOR YOUR COOPERATION

[Otherwise, please continue with the questionnaire]

[Interviewer: If they answered "yes" to any of the questions 21.1-21.6, ask the following, otherwise, SKIP to Q. 25.]

22. Have you had any of the following?

22.1 Head injury that made you lose consciousness? ☐ Yes ☐ No (skip to Q 22.2)

22.1.1 If yes, when did your seizure symptoms start?

☐ Before head injury ☐ Soon after head injury

22.2 Meningitis (brain infection) during childhood? ☐ Yes ☐ No

22.2.1 If yes, when did your seizure symptoms start?

☐ Before an attack of meningitis ☐ Soon after an attack of meningitis

23. What happens to you when you have a seizure or a fit? _____

_____

24. Have you ever hurt yourself when you lose consciousness or during a seizure?

☐ Yes ☐ No

☐ I do not lose consciousness or have seizures (skip to question Q 25)

☐ Cannot remember (skip to question Q 25)

24.1 If yes, how did you hurt yourself?

☐ Fell in the fire ☐ Fell in the water

☐ Fell off your bicycle ☐ Fell while walking along the road

☐ Cut yourself ☐ Other (specify) _____

25. Is there someone in your household with epilepsy or seizures?

☐ Yes, currently is ☐ Yes in the past year, but not currently

☐ Yes, one year or more ago, but not currently ☐ No

25.1 (If yes) Who in your household has epilepsy or seizures? [*check all that apply*]

☐ Mother ☐ Father

☐ Brother/sister ☐ Child

☐ Other relative ☐ Other (specify) _____

(Interviewer: Read the following statement)

Now I want to ask you a few questions about your treatments for *[insert name of symptom or condition they reported having in question 21.1-21.6]*

26. Have you ever consulted a health provider because of this condition?

☐ No (skip to Q 27) ☐ Cannot remember (skip to Q 27)

☐ Yes

26.2 When was the <u>last</u> time you consulted a health provider for your condition?

☐ Within the past month ☐ Within the past year

☐ From one (1) to five (5) years ago ☐ More than five (5) years ago

☐ Can not remember, not sure

26.3 What kind of health provider(s) did you consult and how many times in the past 5 years [*check several boxes if appropriate*]?

☐ A physician _____ times ☐ A neurologist _____ times

☐ A nurse _____ times ☐ A traditional healer _____ times

☐ A psychiatrist/psychologist _____ times

☐ Other (specify _____) _____ times

☐ Can not remember, not sure

26.4 How much did it cost each time you consulted with one health provider [*specify the currency used*]?

☐ A physician _____ ☐ A neurologist _____

☐ A nurse _____ ☐ A traditional healer _____

☐ A psychiatrist / psychologist _____

☐ Other (specify _____) _____

☐ Can not remember, not sure

26.5 How far is the health provider from your house and how did you get there (foot, bicycle, bus, train, taxi, car)?

☐ Physician at _____ km reached by_____ ☐ Neurologist at _____ km reached by____

☐ Nurse at ____ km reached by____ ☐ Traditional healer at ___ km reached by___

☐ Other (specify _____) at _____ km reached by_____

☐ Can not remember

27. Have you ever been hospitalised because of this condition?

☐ No (skip to Q 28) ☐ Cannot remember (skip to Q 28)

☐ Yes Name of Facility _____

27.2 How many times have you been hospitalised in the past 5 years? _____times

27.3 When were you <u>last</u> hospitalised? _____(month) _____(year)

27.3.1 How many days did you stay in hospital? _____ (days)

27.3.2 How much did it cost (specify the currency) _____

27.3.3 How far is the hospital from your house? _____ km

27.3.4 How did you get to the hospital?

☐ By foot/ bicycle ☐ By bus ☐ By taxi

☐ By car ☐ By train ☐ Other (specify) _____

28. Did you ever have any medical tests because of this condition?

☐ No (skip to Q 29) ☐ Cannot remember, do not know (skip to Q 29)

☐ Yes

28.2 What kind of test was it (check as many boxes as appropriate)?

☐ Blood test for cysticercosis ☐ CT scan of the brain

☐ X-Ray of the brain ☐ MRI of the brain

☐ Electroencephalogram (EEG) ☐ Other (please specify) _____

☐ Can not remember, not sure

28.3 When was the last time you had a medical test for this condition?

☐ Within the past month ☐ Within the past year

☐ From one (1) to five (5) years ago ☐ More than five (5) years ago

☐ Can not remember, not sure

28.4 How much did it cost for each test (specify the currency used)?

☐ Blood test for cysticercosis_____ ☐ CT scan of the brain _____

☐ X-Ray of the brain_____ ☐ MRI of the brain _____

☐ Electroencephalogram _____ ☐ Other (specify) _____

☐ Can not remember, not sure

28.5 How far from your house did you have to travel for this test and how did you get there (foot, bicycle, bus, train, taxi, car)?

☐ Blood test for cysticercosis at _____ km at (Facility Name)_____reached by_____

☐ CT scan at _____ km at (Facility Name)_____reached by_____

☐ X-Ray at _____ km at (Facility Name)_____reached by_____

☐ MRI at _____ km at (Facility Name)_____reached by_____

☐ Electroencephalogram at _____ km at (Facility Name)_____reached by_____

☐ Other (specify _____) at _____ km at (Facility Name) _____ reached by_____

☐ Can not remember, not sure

29. Were you ever treated for this condition?

☐ No (the interview is finished) ☐ Can't remember, do not know (interview is finished)

☐ Yes

29.2 When was the last time you used medication for your condition?

☐ Within the past month ☐ Within the past year

☐ From one (1) to five (5) years ago ☐ More than five (5) years ago

☐ Can not remember, not sure

29.3 What medication was it and how many times in the past year did you have to use some (check several boxes if appropriate)?

☐ Phenobarbital _____ times

☐ Dilantin/Tegritol/ Phentoin Sodium _____ times (tick box and underline specific drug name)

☐ Valproic acid _____ times ☐ Traditional medicine _____ times

☐ Other (specify _____) _____ times

☐ Can not remember, not sure

29.4 How much did it cost each time you bought this medication (specify the currency used)?

☐ Phenobarbital _____

☐ Dilantin/Tegritol/Phentoin Sodium _____ (tick box and underline specific drug name)

☐ Valproic acid _____ ☐ Traditional medicine _____

☐ Received for free from health care provider (I did not pay for it myself) _____

☐ Other (specify _____)

☐ Cannot remember, not sure

The following two items should be completed for ALL respondents after direct observation of latrine.

30. Presence and type of latrine (**to be assessed by direct observation**):

☐ Absent ☐ Present and completely enclosed

☐ Present and partially enclosed ☐ Present and open (easily accessible to roaming pigs)

31. Is there evidence of recent use of the latrine (by anyone) (**to be assessed by direct observation**)?:

☐ Yes ☐ No

THIS IS THE END OF THE INTERVIEW

THANK YOU VERY MUCH FOR YOUR COOPERATION.

INTERVIEWER: _____

DATE OF INTERVIEW: _____

Appendix III: Letter of Authorisation for Records Search

MINISTRY OF HEALTH

Telegrams: "MEDICAL", Busia
Telephone:055 22126,/22136
Fax: 055 22136
E-mail: mohbusia@wananchi.com
When replying please quote

Ref No.BSA/

OFFICE OF THE DISTRICT
MEDICAL OFFICER
BUSIA DISTRICT
P O BOX 87
BUSIA (K)
Date.4/06/2005

All Rural Health facilities In charges

Re: Record search by Katherine Ngini in regard to Mental Health Conditions.

This letter serve to introduce Katherine Ngini from the International Livestock Research Institute (ILRI) who is conducting a study on links between porcine cysticercosis and epilepsy.

She would like to examine the OPD mental health records at your facility.

Please accord her the necessary assistance with her enquiries.

MEDICAL OFFICER OF HEALTH
BUSIA DISTRICT

Dr. Mukabi K.J. HSC
District Medical Officer of Health,
Busia District.

Appendix IV: Sample of Case Report Form

Case Report Form for Serology Collection

Patient Information

Last Name:

_____

First Name:

_____

Patient ID No: KDN___ ___ ___

Date of Birth: __ __day/__ __mth/__ __ __ __yr

Sample No: S___ ___ ___
(same as Patient ID No)

Name of person collecting sample:

_____
(printed clearly)

Date Sample Collected: __ __day/__ __mth/__ __ __ __yr

Appendix V: Informed Consent Form – Serology (English and Swahili)

INFORMED CONSENT FORM FOR SEROLOGICAL SAMPLING FOR *TAENIA SOLIUM*

Patient's Name: _____

Patient's Identification Number: _____

Project Title: Assessing the Burden of *Taenia solium* Neurocysticercosis: A Case Study of Busia District

Principal Investigator: Katharine Downie-Ngini

Enumerator: _____

This study is being carried out in collaboration with University of Edinburgh based in Edinburgh, Scotland, UK and the International Livestock Research Institute (ILRI) based in Nairobi, Kenya.

We are asking you to participate in a study to know the importance of the *Taenia solium* tapeworm and neurocysticercosis acquired epilepsy. As you have been identified previously as having symptoms of epilepsy, we would like to take a small sample of your blood from a vein in your arm to confirm if you have epilepsy as a result of the tapeworm found in pork (called *Taenia solium*). In order to do the test successfully, we will need to take approximately 5cc of your blood. This is the same amount as a teaspoon. Your blood sample will be taken by a qualified medical officer or laboratory technician. If you agree to be interviewed and examined, we would also like

to ask you some questions about your health. You are not required to participate in this study and you may leave the study at any time.

Once blood has been collected from you, we will send it to a laboratory in Belgium which is called The Institute of Tropical Medicine in Antwerp, to be tested to determine whether you have been exposed to *Taenia solium* tapeworm. Your blood sample will ONLY be tested for this and nothing else at this time. We will NOT be testing your blood for HIV or AIDS related illnesses. If this test is not conclusive, we may require further tests to be done on the same sample of blood. In view of this, we would like to retain your sample for use in the future to improve the health of your community and to improve methods of finding out whether a person has a pork tapeworm or to better control the pork tapeworm.

Expected benefits: If you are found to be positive for *Taenia solium* either by a blood test or by a CT scan, you will be referred to a qualified medical health practitioner and will be given drugs to treat your condition free of charge. This medical practitioner will explain to you more about your condition and how to complete the treatment effectively so you can be free of epilepsy or cysticercosis related conditions. Everybody who participates in the study will be informed of the outcome of the study. This means that all people who test positively for exposure to *Taenia solium* from their blood sample will be told, those who test negatively for exposure will be informed and those who are given a CT scan will be informed as to whether cysts are present or not.

Statement of confidentiality: Your clinical and laboratory examination and everything that you tell us remains confidential, and you will not be identified by name as a participant in this study. Any information we collect from you will be recorded and identified only by a number, and any publications resulting from this study will not contain your name. Following analysis your blood sample will be destroyed.

In case of complaints: If you experience any problems in relation to your participation in this study you should contact Katharine Downie-Ngini, ILRI, PO Box 30709 00100, Nairobi, Tel: 0204223065.

Investigator's statement: I confirm that I have fully explained to the subject the nature and purpose of the procedures described above and such risks as are involved in its performance. I have asked the subject if he or she has any further questions, and answered these questions to the best of my ability.

Investigator's Signature	Investigator's Name (Print Clearly)
Enumerator's Signature	Enumerator's Name (Print Cearly)

Patient's statement: I understand what has been requested in connection with my participation in this study, and I agree to participate and to give a blood sample. I understand that I may be asked to have a CT scan at a later date and have agreed to this also. I understand that my blood will be taken to a laboratory in Belgium and will only be tested for the purposes outlined in the study and will not at any time by tested for HIV. I do not mind if this sample is used at a later date to further the objectives of this particular study. I understand that if I am a woman between the ages of 15-49 I will be asked to submit a urine sample to determine if I am pregnant or not. If I am pregnant, I will not be asked to participate in the CT scan phase of the study.

I also understand that I may withdraw this consent at any time without any adverse effect.

Date _____

_____	_____
Patient's Signature	Patient's Name (Printed Clearly)
_____	_____
Witness' Signature	Witness' Name (Printed Clearly)

FOMU YA KUONYESHA MAKUBALIANO YA VIPIMO VYA DAMU BAADA YA KUELEWA LENGO LA UTAFITI WA MINYOO AINA YA *TAENIA SOLIUM*

Jina la Mgonjwa: _____

Nambari ya utambulisho ya Mgonjwa: _____

Jina la Mradi: Kuainisha matatizo yatokanayo na Ugonjwa wa Minyoo/Uvimbe katika mishipa ya fahamu (neurone) kati ya wagonjwa wa kifafa katika: Mfano kutoka Wilaya ya Busia

Mtafiti Mkuu: **Katharine Downie-Ngini**

Mtafiti Msaidizi: _____

Utafiti huu unafanyika kwa kushirikiana na Chuo kikuu cha Edinburgh kilichopo Edinburgh, Scotland, Uingereza na shirika la kimataifa la utafiti wa mifugo (ILRI) lililopo Nairobi, Kenya.

Tunakuomba ushiriki katika utafiti huu ili kujua umuhimu wa aina ya minyoo wanaotokana na kula nyama ya nguruwe katika kueneza ugonjwa wa kifafa, minyoo hawa wana fahamika kwa jina la kitaalam kama *Taenia solium*. Kwa kuwa utafiti wa awali umeonyesha kuwa wewe una dalili za ugonjwa wa kifafa, tungependelea kuchukua kiasi kidogo (sampo) cha damu yako kutoka katika mshipa wako wa mkono ili kupima kama una ugonjwa wa kifafa uliotokana na minyoo inayopatikana kwenye nyama ya nguruwe (iitwayo *Taenia solium*). Ili kuweza kupata vipimo sahihi, tutahitaji kutoa CC tano (5cc), hii ni sawa na kijiko kimoja cha chai. Damu hiyo itatolewa na daktari wa binadamu aliyehitimu au mtaalam wa maabara aliyehitimu. Kama utakubali kuhojiwa na kupimwa, tungependa pia kukuhoji maswali machache kuhusiana na afya yako. Kuhusika katika utafiti huu ni kwa hiyari yako, hautalazimishwa hata kidogo na hata baada ya kukubali na kuwa mmoja wa watu wanafanyiwa utafiti, kama utajisikia, kuwa hupendi kuendelea kuhusika unaweza kujitoa wakati wowote.

Baada ya sampo ya damu kutolewa kutoka kwako, itapelekwa katika maabara kufanyiwa uchunguzi kuangalia kama damu yako ina minyoo ya Taenia solium katika maabara nchini Ubelgiji iitwayo " Taasisi ya madawa katika nchi za Tropik" iliyopo katika mji wa Antwerp. Damu hii itapimwa kuwepo kwa minyoo hii tu na sio ugonjwa mwingine wowote. Hatuta tumia damu yako kupima Ukimwi (HIV or AIDS) au magonjwa yoyote yanayohusiana na ukimwi.Tungependa kukuuliza kama uko tayari tutumie damu yako katika siku za baadae katika utafiti wa kutambua kama mgonjwa ana minyoo ya nguruwe na kuendeleza mbinu bora za kupingana na minyoo hao.

Faida zinazotarajiwa kutokana na utafiti huu: Kama ukikutwa na minyoo ya *Taenia solium* kwa njia ya kupimwa damu au au kwa kipimo cha CT scan, utapelekwa kumuona daktari aliyehitimu na utapewa huduma ya matibabu pamoja na dawa bila malipo. Madaktari hawa watakueleza zaidi kuhusu afya yako na jinsi ya kukamilisha matibabu ipasavyo na kupona kabisa kifafa au magonjwa yanayo husiana na uvimbe katika ubongo. Kila atakaye shiriki kwenye utafiti huu ataelezwa matokeo ya utafiti huu. Hii ina maana kwamba watu wote watakaogundulika kuwa na minyoo hii katika damu zao watelezwa, na pia watakao pimwa kwa kipimo cha CT scan wataelezwa kama kuna uvimbe katika ubongo wao au hakuna.

Tamko la kuhufadhi siri: Afya yako na matokeo ya vipimo vyako katika maabara (Laboratory examination) na chochote ambacho umetueleza kitabaki kuwa siri. Hatutatumia jina lako mshiriki katika utafiti huu. Kila maelezo tutakayo pata kutoka kwako tutayaandika na kuyatambua kwa nambari tu, na matokeo yoyote katika utafiti huu hayatakuwa na jina lako. Mara baada ya utafiti huu sampo yako ya damu itaharibiwa na haitatumika tena kwa dhumini lingine lolote.

Kama utakuwa na malalamiko au matatizo yoyote: Endapo utakumbana na matatizo yoyote kuhusiana na ushiriki wako katika utafiti huu tafadhali wasiliana na Katharine Downie-Ngini, ILRI, PO Box 30709 00100, Nairobi, Simu no 0204223065.

Tamko toka kwa Mtafiti: Nina uhakika nimemueleza muhusika kiundani nia na madhumuni pamoja na mambo yanayoambatana na utafiti huu. Pia nimemweleza madhara yanayoweza kujitokeza wakati wa utafiti. Nimempa muhusika nafasi ya kuuliza maswali kuhusiana na uafiti huu na nimeyajibu kadri ya uwezo wangu.

_____	_____
Sahihi ya Mtafiti	Jina la Mtafiti (Liandikwe kihusahihi)
_____	_____
Sahihi ya Msaidizi wa mtafiti	Jina la Msaidizi wa mtafiti (Liandikwe kihusahihi)

Maelezo ya Mgonjwa: Nimeyaelewa vyema maelezo kuhusiana na utafiti huu na mambo ninayohitajika kufanya kama mshiriki katika utafiti huu, ninakubali kutoa kiasi kidogo cha damu (sampo) itakayo tumika katika utafiti huu. Ninaelewa kwamba nitahitajika kuchukuliwa kipimo cha CT scan hapo baadae. Nimeelewa kwamba damu yangu itatumika tu kwa madhumuni yalioelezwa katika utafiti huu na kwa wakati wowote haita tumika kupima ukimwi (HIV or AIDS). Ninaruhusu sampo ya damu yangu itumike hapo baadae kwa madhumini ya utafiti huu. Ninaelewa kwamba kama mimi ni mwanamke mwenye umri kati ya miaka 15-49 nitahitajika kutoa sampo ya mkojo kuangalia kama nina mimba au la! Kama nina mimba, sitahitajika kushiriki katika vipimo vya CT scan kama sehemu ya utafiti. Na pia naelewa kwamba ninaweza kujitoa wakati wowote. bila matatizo.

Tarehe _____

_____	_____
Sahihi ya Mgonjwa	Jina la Mgonjwa (Liandikwe kihusahihi)
_____	_____
Sahihi ya Shahidi	Jina la Shahidi (Liandikwe kihusahihi)

Appendix VI: Informed Consent Form - CT Scan

INFORMED CONSENT: CT Scan

Instructions for Obtaining Informed Consent

Fieldworker: Identify yourself and introduce the purpose of the survey by reading from the following script (this script appears at the beginning of each survey packet for your convenience):

Invitation and Purpose:

"Hello and good morning/afternoon. My name is _____ and I am here to ask if you would mind participating in a CT scan which is a follow-up from our previous survey which took place in December of last year."

(If patient is a woman of reproductive age, she must provide urine for a pregnancy test).

I would like first of all to ask you to give us some of your urine so that we can test to see if you are pregnant. If you are pregnant, you will not be asked to go for a CT scan (If not pregnant or male, proceed as follows).

Following the testing of the blood and the determining of whether or not you have been exposed to *Taenia solium* and have antibodies to *Taenia solium* present in your blood, we will ask some of you (both positive and negative) to undergo a CT scan to determine whether there may be any cysts in your

brain. This scan is not painful and simply involves a machine taking a picture of your brain to see if there are any cysts present. Cysts in your brain may be a cause of your epilepsy. If there are cysts present, we will be able to send you for treatment for these cysts.

Please remember that you do not have to participate in this study if you do not want to. Also, if you have any questions, please ask.

All information, including your name and your health status (whether you have cysts or have been exposed to the pork tapeworm) will be kept confidential by everyone participating in this study. We will also let you know whether you have cysts or not, or whether you have been exposed to the tapeworm.

Procedures:

You will be taken, along with others participating in this study, to the Aga Khan Hospital in Kisumu so that a machine can take a picture of your brain. Computed tomography (CT) is a diagnostic procedure that uses special x-ray equipment to obtain cross-sectional pictures of the body. The CT computer displays these pictures as detailed images of organs, bones, and other tissues. This procedure is also called CT scanning, computerized tomography, or computerized axial tomography (CAT).

During a CT scan, the person lies very still on a table. The table slowly passes through the center of a large x-ray machine. The person might hear whirring sounds during the procedure. People may be asked to hold their breath at times, to prevent blurring of the pictures. You may be asked to let the doctor inject some dye into your vein in order that different parts of your brain show up clearly on the scan. This injection will be administered by qualified health personnel.

Having your picture taken does not hurt at all and will be carried out by a trained medical health professional. The information we obtain regarding your scan will be shared with you but kept confidential from others participating. Again, you are under no obligation whatsoever to participate in this study. If you have any questions about this, please ask them now.

Risks, Discomforts and Inconveniences:

We estimate that the trip to Kisumu and the scan will probably take the entire day. We will drive you there and bring you back home.

Some people may be concerned about the amount of radiation they receive during a CT scan. It is true that the radiation exposure from a CT scan can be higher than from a regular x-ray. However, *not* having the procedure can be more risky than having it, especially if cancer is suspected. People considering CT must weigh the risks and benefits.

Computed tomography scans do not cause any pain. However, lying in one position during the procedure may be slightly uncomfortable. The length of the procedure depends on the size of the area being x-rayed; CT scans take from 15 minutes to 1 hour to complete. For most people, the CT scan is performed on an outpatient basis at a hospital or a doctor's office, without an overnight hospital stay.

Benefits:

The benefits to having this scan done are that if we detect cysts in the picture that has been taken, you will be referred for treatment afterwards.

Privacy and Confidentiality:

The information gathered in the interview will be kept confidential and will not be shared with any persons or agencies not affiliated with this study. The results of your scans will be combined with others who participate in the study in such a way that it is not possible to associate these responses with others. You will be assigned a code, and this code will be stored separately

from the results of the survey. Individual responses will thereafter be referred to by codes alone. The names of household members will not be shared with individuals outside the survey organization and the identifying information linking names to codes will be destroyed once the information is compiled into a computer

INFORMED CONSENT FORM

To be completed PRIOR to conducting interview. Interview CANNOT take place unless this form is completed.

INTERVIEWER'S STATEMENT

_____ has been informed of the nature and purpose of the procedures described above including any risks involved in its performance. He or she has been given time to ask any questions and these questions have been answered to the best of the investigator's ability. A signed copy of this consent form will be made available to the subject.

_____
Investigator's Signature

_____ _____
Investigator's Name (printed clearly) Date

SUBJECT'S ORAL OR SIGNED CONSENT

I have been informed about this research study, its possible benefits, risks, and discomforts. I hereby agree to take part in this research as a subject. I recognize that my participation is voluntary and that I am free to withdraw this consent and quit this project at any time, and that doing so will not cause me any penalty or loss of benefits that I would otherwise be entitled to enjoy. I may also skip any question that I do not wish to answer.

_____ _____

Subject's Signature (or Print) Date
(Not needed if oral consent is given)

_____ _____

Witness to Oral Consent Date

_____ _____

Subject's Legal Representative Date
(if applicable)

Appendix VII: Enumerators' Confidentiality Agreement (English and Swahili)

CONFIDENTIALITY AGREEMENT:

To be completed PRIOR to collecting serology sample or conducting interviews. Neither of these two actions can take place unless this form is completed.

To be completed by the following personnel: (tick one)

Clinical Officer _____

Lab technician _____

Enumerator _____

I, _____ understand that all information and data gathered from subjects or patients during this study shall be kept confidential. This means that I shall not discuss any information or results emanating from this study with anyone, including colleagues who have participated in the study. A signed copy of this confidentiality agreement will be made available to the subject.

_____

Investigator's Name

_____

Investigator's Occupation (i.e., enumerator, lab technician, etc.)

_____

Investigator's Signature

_____

Date

MKATABA WA SIRI NA MTAFITI MSAIDIZI

Mkataba huu unatakiwa kujazwa **KABLA** ya kuchukua vipimo vya damu au kuwauliza wananchi maswali. Hakuna kati ya mambo haya miwili linaloweza kufanyika kabla mkataba huu haujasainiwa.

Kujazwa na wafanyakazi wa utafiti: (chagua moja)

Daktari wa binadamu/mtabibu: _____

Mtaalamu wa maabara (laborator _____

Mtafiti msaidizi: _____

Mimi_____, ninaelewa kuwa maswala yoyote yanayowahusu wananchi watakaofanyiwa utafiti, ikiwa ni pamoja na majibu watakayotoa kufuatia maswali tutakayowauliza na matokeo yote ya vipimo tutakavyochukua yatakuwa siri. Hii ina maana kwamba mimi sitamwambia mtu yeyote jambo lolote kuhusiana na matokeo ya utafiti huu hata kama ni wananchi wengine waliohusika kwenye utafiti huu (wagonjwa wengine). Nitaweka sahihi kwenye mkataba huu na kumpatia kila mwananchi atakayefanyiwa utafiti.

_____	_____
Jina la Mtafiti	Kazi au cheo cha Mtafiti (kama vile daktari mtabibu, mtaalamu wa maabara, n. k)
_____	_____
Sahihi ya mtafiti	Tarehe

Appendix VIII: Grant Award Letter

ANIMAL HEALTH PROGRAMME

PROJECT MEMORANDUM FORM

AHP Project Number:
(to be assigned by AHP)

AHP-BO-05/05

Project Title:

Assessing the burden of cysticercosis, with relevance to both human health and livestock production economics, on smallholder communities in western Kenya

Abbreviated Title: (max. 30 characters)

Cysticercosis in Western Kenya

The completed form should be submitted to **The Programme Manager, Animal Health Programme, Centre for Tropical Veterinary Medicine, The University of Edinburgh, Easter Bush Veterinary Centre, Roslin, Midlothian EH25 9RG** as a signed hard copy.

DFID Department for International Development

THE UNIVERSITY OF EDINBURGH

AHP Animal Health Programme DFID

Each section contains headings only. You should expand the space under each heading to suit your text. You may expand the tables or forms contained, if necessary, but not alter their format.

SECTION A: ADMINISTRATIVE INFORMATION

Please fill in the unshaded sections. Shaded sections are for office use only.

Date Application Received	
R Number	**AHP-BO-05/05**

Project Leader	A. Lee Willingham, DVM, PhD
Applicant Institution (registered offices)	International Livestock Research Institute
Contracts Manager/ Finance Officer of applicant institution	Brigitte Laude
Collaborators and collaborating institutions	Katharine Downie-Ngini, ILRI, Nairobi, Kenya Dr Thomas Randolph, Human Health Impacts, ILRI, Nairobi, Kenya Stella Massawe, Targeting Pro-Poor Interventions, ILRI, Kenya Dr Samuel Githigia, Veterinary Faculty, University of Nairobi, Kenya Florence Mutua, Veterinary Faculty, University of Nairobi, Kenya Dr Erastus Amayo, Medical Faculty, University of Nairobi, Kenya Dr Eric Fèvre, CTVM, University of Edinburgh, Scotland Dr Sue Welburn, CTVM, University of Edinburgh, Scotland

Full Title	Assessing the burden of zoonotic disease (cysticercosis) with relevance to both human health and livestock production economics, on smallholder communities in western Kenya
Country Focus	Kenya
Project Location	Busia and other western districts, Nairobi
Names of co-funders (if relevant)	n/a

Cost to AHP Research Programme	£20,000.00

Appendix IX: Supplementary Risk Factor Questions

Last name : _____ First Name : _____

If Child then : Patient number : _____

Father's Name :_____ District _____

Division: _____

Mother's Name:_____ Location: _____

Sub-location: _____

Village _____

Hut (house) number _____

How long have you lived in this village?____ (yrs.)

GPS Reading North:_____(Format 00.xxxxx)

East:: _____(Format 00.xxxxx)

Altitude: _____(Format xxxx m)

1. How old are you?_____ (years)
2. What is your date of birth?____ Day_____Month_____ Year
3. Sex ☐ Male ☐ Female

14. Do you keep pigs?

☐ Yes ☐ No (Skip to Q 15)

14.1 What type of pigs do you keep?

☐ Foreign ☐ Native

☐ Both foreign and native ☐ Can not remember, do not know

14.2 Of the pigs that you have, how many are for? [*read each choice and record the number*]

Home consumption _____ Trading _____

Reproduction _____ Other (specify): _____

14.3 How do you keep your pigs?[*read questions 14.3.1 to 14.3.4 one after the other*]

14.3.5 During the planting season

☐ In a pen ☐ Free ranged

☐ Tethering ☐ Other (specify): _____

14.3.6 During the growing season

☐ In a pen ☐ Free ranged

☐ Tethering ☐ Other (specify): _____

14.3.7 During the harvesting season

☐ In a pen ☐ Free ranged

☐ Tethering ☐ Other (specify): _____

14.3.8 During the fallowing season

☐ In a pen ☐ Free ranged

☐ Tethering ☐ Other (specify): _____

14.4 What do your pigs eat? [*Check all that apply.*]

☐ Pasture ☐ Kitchen left overs

☐ Commercial feeds ☐ Other (specify): _____

14.5 How often do you slaughter pigs at home?

☐ At least once a month ☐ Less than once a month but at least once a year

☐ Less than once a year ☐ Never (Skip to Q 14.6)

☐ Can not remember, do not know (Skip to Q 14.6)

14.5.1 If ever, how often was the meat inspected by a meat inspector?

☐ Always ☐ Almost always ☐ Sometimes

☐ Never☐ Can not remember, do not know

14.6. What price do you usually sell your pigs when they are ready to be slaughtered (specify the currency used, this can be money or barter)?_____ (skip to Q 32)

14.7. What price do you usually sell your piglets (aged 4 months or less) (specify the currency used, this can be money or barter)? _____(skip to Q 32)

15. Have you ever owned pigs? *[If they answer "yes", ask when they owned pigs]*

☐ Yes, in the past year ☐ Yes, one (1) to five (5) years ago

☐ Yes, more than five (5) years ago ☐ No (skip to Question 32)

15.1. What kind of pigs were they?

☐ Foreign ☐ Native

☐ Both foreign and native ☐ Can not remember, do not know

32. Do your neighbours keep pigs currently?

☐ Yes ☐ No

33. Have your neighbours or people close to you in the village kept pigs in the last five years?

☐ Yes ☐ No

34. Do pigs ever come on your compound?

☐ Yes ☐ No

21.5 Were you ever told that you had epilepsy or that you had had an epileptic seizure?

☐ Yes, currently has ☐ Yes in the past year, but not currently

☐ Yes, one year or more ago, but not currently ☐ No (*skip to question 21.6)*

☐ Can not remember, do not know

21.5.1. If yes, then by whom?

☐ Health professional ☐ Traditional Healer

☐ Relative ☐ Friend, neighbour

21.6 Have you ever had seizures or fits?

☐ Yes, currently has ☐ Yes in the past year, but not currently

☐ Yes, one year or more ago, but not currently

☐ No *(skip to question 18)* ☐ Can not remember, do not know *(skip to question 18)*

21.6.1 How often has this happened?

☐ Only once ☐ More than once

21.6.2 How old were you when this first happened? _____ years

18. Have you ever heard of tapeworm infection in humans?

☐ Yes ☐ No *(Skip to question 19)*

18.1 How did you learn about it?

☐ By a doctor ☐ By a friend or family member

☐ By a traditional healer ☐ On the radio / newspaper

☐ Other (Specify)_____

18.2 How does a person know if they have a tapeworm?

☐ They can see it in their faeces ☐ They have diarrhea

☐ They have fever ☐ Other Specify)_____

☐ I don't know

18.3 Have you ever had a tapeworm or seen small parts (segments) of worms in your faeces? (*Show photographs of proglottids*)

☐ Yes ☐ No *(skip to q 18.4)*

☐ I don't know/can not remember *(skip to q 18.4)*

18.3.1 When that happened, what did you do? [*check all that applies*]

☐ Went to a primary health care provider (hospital, clinic, dispensary)

☐ Went to the pharmacy to get a drug to treat it

☐ Went to a traditional healer ☐ Did nothing

☐ I can not remember, I do not know

18.4 How does a person get tapeworm infection?

☐ They do not wash their hands ☐ They eat undercooked pig meat

☐ They are in contact with an infected person

☐ Other (Specify) _____

☐ I don't know

36. Has anyone in your household had a tapeworm infection?

☐ Yes, currently has ☐ Yes in the past year, but not currently

☐ Yes, one year or more ago, but not currently

☐ No (skip to Q 22) ☐ Can not remember, do not know

Appendix X: Asset Index

	Very Poor (VP)	√	Not so Poor (NSP)	√	Non-poor (NP)	√
Cooking Utensils	Leaking or damaged Very few		Better pots, 1 set of dishes 1 set of dishes		Good quality More than one set of dishes	
Sleeping Conditions	Floor or mat		Mattress		Bed	
Clothes	Torn or dirty		Patched		Good clothes	
Slippers	No footwear		Repaired		Better shoes	
	Odd shoes or damaged		Second hand		New shoes or slippers	
Food	Cassava or millet meal, 1 meal a day		Cassava, millet meal or maize meal with a vegetable (kunde, sukuma etc), 2 meals a day		Ugali, rice, millet meal or cassava with a vegetable, meat or fish, 3 meals a day	
Health Condition	Skin rash, infected eyes, sore feet, cough, running nose, diarrhoea		Some of the same as VP but better health and buys medicines from duka or kiosk		Visits the doctor, buys medicines from a pharmacy	

	Very Poor (VP)	√	Not so Poor (NSP)	√	Non-poor (NP)	√
Schooling	No children in school		Boy children only in school		All children in school	
Housing	Thatch roof		Part of roof is mabati		All of roof is mabati	
	No door		Solid front door		Two doors	
	Cloth to cover entrance way		One room cement floor		All of house is cement floor	
Furniture	No chairs		2 chairs or stools		Sufficient furniture	
	Some benches		table			
Utilities	No toilet		Shallow pit latrine		Good enclosed pit latrine	
	One lantern		2 lanterns		Sufficient lights	
	Only has fire for light		River for water		Well for water	
Domestic Employees	none		none		1 employee	

	Very Poor (VP)	√	**Not so Poor (NSP)**	√	**Non-poor (NP)**	√
Transportation	Walking		Paying for transport		Owns bicycle	
					Owns motorcycle	
Radio	none		Old or damaged		Good or new	
Animals	none		Sheep, chickens		Cows, sheep	
Economic Activities	No income or working as a labourer without pay		Part time paid employment		Regular paid employment	
Business Income	Gathering and selling items like nuts and firewood		Buying and selling firewood, market activities		Larger participation in market, permanent stall, shop etc	

Appendix XI: Investigators' Signatures

ILRI
INTERNATIONAL LIVESTOCK RESEARCH INSTITUTE

Katharine Downie-Ngini
Centre for Infectious Diseases
College of Medicine and Veterinary Medicine
University of Edinburgh
EH25 9RH
UK

This document is to certify that having read the Ethical Protocol submission "Communities and Zoonoses: A Case Study of Busia District", I agree to act as the principal investigator in this study.

Katharine Downie-Ngini _____ Date _____

CENTRE FOR INFECTIOUS DISEASES
incorporating
Centre *for* Tropical Veterinary Medicine
College of Medicine and Veterinary Medicine
The University of Edinburgh
Scotland UK
EH25 9RG

Telephone +44 (0) 131 650 6287
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Email: ian.maudlin@ed.ac.uk
http://www.vet.ed.ac.uk/ctvm

Sue Welburn
Professor of Medical and Veterinary Molecular Epidemiology
Centre for Infectious Diseases
College of Medicine and Veterinary Medicine
University of Edinburgh
EH25 9RH
+441316506228

This document is to certify that having read the Ethical Protocol submission "Communities and Zoonoses: A Case Study of Busia District", I agree to act as an investigator in this study.

_____

Sue Welburn Date 23.11.06

A. Lee Willingham III, DVM, PhD
WHO/FAO Collaborating Center for Parasitic Zoonoses,
Royal Veterinary and Agricultural University
Dyrlaegevej 100 1870
Frederiksberg,
DENMARK
tel: +45 35282775
fax: +45 35282774
e-mail: awi@kvl.dk

This document is to certify that having read the Ethical Protocol submission "Communities and Zoonoses: A Case Study of Busia District", I agree to act as an investigator in this study.

A. Lee Willingham III Date

Erastus Olonde Amayo, MB, ChB, MMed (Internal Medicine)
Associate Professor
Department of Clinical Medicine and Therapeutics
University of Nairobi
PO Box 19676 00100
Nairobi
KENYA

This document is to certify that having read the Ethical Protocol submission "Communities and Zoonoses: A Case Study of Busia District", I agree to act as an investigator in this study.

_____ Date _____

Erastus Olonde Amayo